# Assessment of strength and power responses to resistance exercise in young and middle-aged trained males

Thesis submitted in accordance with the requirements of the University of Chester for the degree of Doctor of Philosophy

By John F. T. Fernandes

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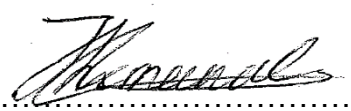
This work is original and has not been previously submitted in support of a Degree of Doctor of Philosophy or other course.



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**Abstract**

Little is known about the muscle function capabilities of trained middle-aged males and how they differ to younger counterparts. Accordingly, the overall aim of the research documented in this thesis was to compare the acute muscle function responses to resistance exercise in middle-aged and young resistance trained males. The first study (Chapter 3) examined the intra- and inter-day reliability of an ecologically valid device (FitroDyne rotary encoder) for measuring upper and lower-body muscle function during three popular multi-jointed resistance training exercises (bench press, squat, and bent-over-row), and confirmed that it was capable of detecting moderate changes in muscle function across a range of submaximal loads. In the second study (Chapter 4) the load-velocity and load-power relationships were investigated during the same exercises among 20 young (age 21.0 ± 1.6 y) and 20 middle-aged (age 42.6 ± 6.7 y) resistance trained males, and it emerged that, despite their regular training, the middle-aged males were unable to achieve velocities at low external loads and peak powers at all external loads as high as the young males across a range of external resistances.

Study three (Chapter 5) proceeded to compare the internal (heart rate (HR), OMNI-ratings of perceived exertion (RPE) and sRPE) and external (peak velocity and power and volume load) loads experienced during high volume squatting exercise, and the fatigue responses among nine young (age 22.3 ± 1.7 years) and nine middle-aged (age 39.9 ± 6.2 years) resistance trained males. The findings highlighted that internal, but not certain markers of external (peak power and volume load), load responses can be monitored during exercise in a like manner between these age groups. Moreover, compared to young resistance trained males, middle-aged males can expect greater decrements in peak power after lower-limb resistance exercise. In

the final study (Chapter 6), the time-course of recovery in nine trained young (age 22.3 ± 1.7 years) and nine trained (39.9 ± 6.2 years) and nine untrained (44.4 ± 6.3 years) middle-aged males after high volume lower-body resistance (muscle damaging) exercise was investigated. Of practical importance, it emerged that compared to the young males, the trained middle-aged males experienced greater symptoms of muscle damage and an impaired recovery profile, the implication of which is the need for trained middle-aged males to adopt strategies to enhance their recovery. Furthermore, both middle-aged groups experienced similar symptoms of muscle-damage, albeit the untrained group demonstrated greater losses in peak power at low and high external loads.

For the first time, the current research has determined that middle-aged males, despite regular resistance training, are subject to losses in peak velocity and power output across a range external loads, compared to young males. When undergoing lower-body resistance training to ameliorate these decrements, applied practitioners can use internal load markers and peak velocity, but not peak power or volume load, to monitor trained young and middle-aged males alike. Furthermore, the muscle damage response (24 to 72 h), and losses in peak power (0 to 72 h), after lower-body resistance exercise are greater in trained middle-aged than young males. Consequently, future research should seek to corroborate these observations in upper-body exercise and determine the effectiveness of strategies (e.g. nutritional intake) to enhance recovery in middle-aged males.

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"…keep your nose out the sky, keep your heart to God and keep your face to the rising sun" Kanye West, Family Business

**Publications**

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# 1. Introduction

## 1.1 Ageing and muscle function

The ageing process is associated with declines in athletic capability or performance. For example, Pantoja and colleagues (2016) recently reported a ~1% decline per year between the ages of 39 and 96 years in maximal velocity and power outputs during a 30 m sprint. Baker and Tang (2010) also noted a 25% difference in weightlifting performance (World Records) between the 30- and 60-year old age categories. These reductions in performance are particularly challenging for the growing number of middle-aged male athletes (Lepers et al., 2013; Tanaka & Seals, 2008), many of whom strive to maintain or improve their athletic capability as they get older.

The age-associated losses in muscle mass (sarcopenia; Narici, Reeves, Morse & Maganaris, 2004; Welle, 2002) and strength and power (dynapenia; Candow & Chilibeck, 2007; Clark & Manini, 2012; Izquierdo et al., 1999) are likely to be a major cause of the aforementioned decline in athletic performance as athletes get older. Though early work by Frontera, Hughes, Lutz and Evans (1991) suggested that sarcopenia was the major determinant of dynapenia, more recent longitudinal and cross-sectional research indicates that it cannot fully account for the losses of strength and power as people age (Delmonico et al., 2009; Goodpaster et al., 2001; Hughes et al., 2001; Petrella et al., 2005; Visser et al., 2000). The loss of power with ageing appears to be greater than the loss of strength (Delmonico et al., 2009; Izquierdo et al., 1999; Power, Dalton, Rice & Vandervoort 2012; Skelton, Greig, Davies & Young, 1994; Thom, Morse, Birch & Narici, 2005) which, in terms of a variety of sporting tasks, will impact on performance achievements (Baker, 2001a; Baker, 2001b; Cronin & Hansen, 2005; Delaney et al., 2015; Lopez-Segovia, Dellal, Chamari & Gonzalez-Badillo, 2014; Loturco et al., 2016a; Loturco et al., 2016b; Sleivert & Taingahie, 2003).

In addition, these age-associated changes in muscle mass, strength and power do not appear to be uniform. For example, a variety of studies report greater losses in muscle mass (Abe et al., 2011; Abe, Loenneke, Theibaud, & Fukunaga, 2014a; Candow & Chilibeck, 2005; Janssen, Heymsfield, Wang & Ross, 2000), strength and power (Candow & Chilibeck, 2005; Frontera et al., 2000; Lynch et al., 1999; Noguiera et al., 2013) in the lower- compared to the upper-body. The mechanisms for these site-specific losses are unclear but it is speculated that during daily living lower-body movements are supplemented by upper-body contributions (as when using the upper-body to rise from a chair; Macaluso & DeVito, 2004) and the lower-body undergoes more severe changes in muscle contractile units (e.g. decreases in the specific tension of type 1 and 2 fibres; Larsson, Li & Frontera, 1997) and other tissues (e.g. increases in fat and connective tissue; Lynch et al., 1999; Overend, Cunningham, Paterson & Lefcoe, 1992). However, more work is needed to gain an understanding of upper- and lower-body muscle functional characteristics of resistance trained middle-aged males and how they compare to younger males. A deeper understanding of these muscle function characteristics might also inform resistance training practices in middle-aged athletes that could offset the general age-related changes observed.

## 1.2 Internal and external loads with ageing

Resistance training is a method of physical conditioning that can enhance muscle function characteristics (typically strength, hypertrophy or power) when used longitudinally. There is mounting evidence indicating that gains in muscle mass, strength and power can be made in older populations (Bottaro, Machado, Nogueira & Veloso, 2007; Kongsgaard, Backer, Jorgensen, Kjaer & Beyer, 2004; Kosek, Kim, Petrella, Cross & Bamman, 2006; Newton et al., 2002; Roth et al., 1999; Sayers &

Gibson, 2010; Sayers & Gibson, 2014; Straight, Lindheimer, Brady, Dishman & Evans, 2016). Importantly, the success of resistance training is predicated on the ability of the athlete (or coach) to quantify and regulate the stress imposed (Scott, Duthie, Thornton & Dascombe, 2016). If the training load is too high, injury can occur, whereas insufficient stress might prevent neuromuscular adaptation (Foster, 1998; Gabbett, 2016; Halson, 2014). Consequently, applied practitioners need to measure a variety of *internal* (i.e. HR, RPE) and *external* (i.e. velocity, power) load variables to quantify the training load. However, there is no wholly agreed marker used to monitor resistance training (McGuigan & Foster, 2004; Scott et al., 2016; Sweet, Foster, McGuigan & Brice, 2004) due to the numerous variables (e.g. velocity, rest time, repetitions, volume load; Schoenfeld, 2010) that can invoke a resistance training adaptation.

When monitoring the resistance training of middle-aged adults, it is apparent that internal load markers are different to those of young adults. For example, in young (~21 to 28 years), compared to older individuals (~57 to 84 years), there are reports of higher HRs (Smolander et al., 1998) and blood lactate concentrations (Smilos et al., 2007; Walker et al., 2013), and lower (Allman & Rice, 2003; Justice et al., 2014), higher (Pincivero, 2010) and similar (Pincivero et al., 2010) RPEs after resistance exercise (i.e. an activity performed against a load). However, there are no differences in absolute or relative HRs (Kawano et al., 2008), blood lactate concentration and RPE (Manini et al., 2012) when resistance training is performed to failure in young (~21 to 28 years) and older (~48 to 67 years) males. Moreover, while external load appears to drive resistance training adaptations (Schoenfeld, 2010), no study has compared the external load experienced during resistance exercise in young and middle-aged men.

## 1.3 Fatigue and ageing

Resistance training may induce fatigue (i.e. an inability to maintain the expected force or power output; Edwards, 1981), but the nature of this response as athletes age is unclear. For example, recent meta-analyses have reported that ageing is associated with an enhanced fatigue resistance after isometric, but not dynamic contractions (Avin & Frey Law, 2011; Christie, Snook & Kent-Braun, 2011). Furthermore, when the losses of velocity and power are used as the markers of fatigue, older (~64 to 75 years) males experience greater fatigue than their young (~27 years) counterparts during knee extension (Dalton, Power, Vandervoort & Rice, 2012; Dalton, Power, Paturel & Rice, 2015; Petrella, Kim, Tuggle, Hall & Bamman, 2005), but not sit-to-stand exercise (Christie et al., 2011; Petrella et al., 2005). Such findings (i.e. the greater fatigue in older males) are likely owing to the age-related slowing of muscle contraction during knee extension exercise (Dalton et al., 2012; Petrella et al., 2005), whereas differences in fatigue rates for knee extension and sit-to-stand exercise have been attributed to task specificity (Petrella et al., 2005). That is, both age groups are used to performing sit-to-stand tasks, but not knee extension movements (Petrella et al., 2005). Importantly, knee extensions and sit-to-stand tasks might not reflect the multi-jointed compound movements used by the middle-aged male who regularly resistance trains. As such, a study that examines the fatigue response involving such movements would be both innovative and informative to such an athlete.

## 1.4 EIMD and ageing

Another consequence of resistance exercise, particularly when it comprises high-volume or eccentrically biased muscle actions is damage to the muscle ultrastructure (Hortobagyi et al., 1998; Roth et al., 1999). The consequences of exercise-induced

muscle damage (EIMD) include muscle sarcomere disruptions and ensuing membrane damage (e.g. Damas, Nosaka, Libardi, Chen & Ugrinowitsch, 2016; Hyldahl & Hubal 2014; Morgan 1990; Morgan & Allen 1999; Proske & Morgan 2001). During eccentric muscle contractions, lengthening is non-uniform and weaker sarcomeres extend beyond their myofilament overlap and fail to re-interdigitate (Morgan, 1990; Morgan & Allen, 1999; Proske & Morgan, 2001). This is attributed to an increased stress per myofibre that is consistent with eccentric contractions (Enoka, 1996) and is known as the 'popping-sarcomere hypothesis' (Morgan, 1990; Morgan & Allen, 1999; Proske & Morgan, 2001). Thereafter, a loss of calcium homeostasis leads to excitation-contraction (E-C) coupling dysfunction and a prolonged loss of muscle strength and other associated symptoms (Damas et al., 2016; Hyldahl & Hubal, 2014; Morgan, 1990; Morgan & Allen, 1999).

The symptoms of EIMD include increases in muscle soreness, intramuscular enzymes in the blood serum and plasma, and of most importance to the athlete, impairments in muscle function that include reductions in muscle strength and power (Damas et al., 2016; Flavo & Bloomer, 2006; Hyldahl & Hubal, 2014). Typically, these symptoms peak between 24 and 48 hours after the initial bout and are recovered (i.e. returned to baseline values) by seven days (Damas et al., 2016; Flavo & Bloomer, 2006; Hyldahl & Hubal, 2014). The symptoms are highly individualised, not synchronous (Clarkson et al., 2005; Hubal, Rubenstein, Clarkson, 2007; Machado & Willardson, 2010), and are affected (reduced) in subsequent bouts where prior eccentric exercise has occurred up to six months before, for example, in the elbow flexors (Hyldahl, Chen & Nosaka, 2017; Nosaka, Sakamoto, Newton & Sako, 2001). This protective adaptation is known as the 'repeated bout effect' (RBE; McHugh, 2003; Hyldahl et al., 2017) and appears to be less pronounced in older compared to younger

men (Lavender & Nosaka, 2006b; Gorianovas et al., 2013). It is plausible, therefore, that middle-aged men who resistance train will exhibit comparable symptoms of EIMD to their untrained counterparts.

The available data comparing recovery from EIMD between age groups is equivocal. Work by Lavender and Nosaka (2006a; 2007) has reported greater symptoms of EIMD in young (~20 years) compared to older males (~71 years), while other studies have observed greater EIMD in older (~59 to 66 years) compared to younger (~20 to 30 years) adults (Manfredi et al., 1990; Ploutz-Snyder, Giamis, Formikell & Rosenbaum, 2001). However, after resistance exercise some research has reported comparable symptoms of EIMD in young (~19 years) and middle-aged (~48 years; Gordon et al., 2017; Lavender & Nosaka, 2008) or even older males (~64 to 76 years; Buford et al., 2014; Chapman, Newton, McGuigan & Nosaka, 2008; Clarkson & Dendrick, 1988; Roth et al., 1999). To date, only two studies (Buford et al., 2014; Gordon et al., 2017) have investigated the effects of lower-body resistance exercise on recovery profiles between age groups. Buford et al. (2014) reported that recovery from muscle damaging unilateral plantar flexor exercise was similar between young (~23 years) and older (~76 years) physically active adults, when controlling for general physical activity (i.e. any activity that requires energy expenditure). Similarly, in recreationally trained young (~22 years) and middle-aged (~47 years) males, Gordon and colleagues (2017) observed no differences in indirect markers of EIMD after muscle damaging knee extensor exercise. Indeed, Gordon et al. (2017) recommend that future studies might adopt more ecologically valid muscle damaging protocols, for example, multi-jointed resistance exercises, that middle-aged males might perform in order to help prolong their athletic careers.

### 1.5 Aims of the thesis

The literature on ageing is typically health-related and focuses predominantly on people older than 60 years. As such, the muscle function (i.e. strength, velocity and power) characteristics of trained middle-aged males are largely unknown. Moreover, if middle-aged males demonstrate impairments in muscle function then resistance training might provide a means of compensating for such changes. However, there is a dearth of research regarding the acute changes in muscle function that occur after resistance training in such athletes. Consequently, the aims of current research were to establish a reliable measurement tool (Chapter 3) for determining the load-velocity and load-power characteristics in trained middle-aged males (Chapter 4), and to examine the subsequent fatigue and recovery responses from high volume squatting exercise (Chapter 5 and 6, respectively).

# 2. Review of literature

## 2.1 An introduction to ageing

World population predictions reach as far as the year 2100 where the population is estimated to be 11.18 billion (Department of Economic and Social Affairs, 2017). Alongside these population increases are other demographic transformations, such as a longer life expectancy. As Ferricci and colleagues (2008) indicate, population ageing is a global occurrence. In 2015 only 14% of the world population was aged 60 years and over; by 2050 and 2100 this number is suggested to reach 26 and 36.7%, respectively (Department of Economic and Social Affairs, 2015). Similarly, the number of middle-aged people (30 to 59-year-old) in the U.K., and globally, is increasing (Office for National Statistics, 2014) with the expectation that the number will rise from 15.7 million in 2014 to 16.3 million in 2020. Improvements in medical care, a decline in the leading causes of mortality and a greater appreciation for factors that enhance longevity are said to contribute to these demographic transformations (Baker & Tang, 2010; Ferrucci et al., 2008).

Concomitant with this transformation is a growing number of middle-aged athletes, for example the 25% increase between 1986 and 2010 in ‘Master’ athletes competing in the ultra-endurance sports (Lepers et al., 2013; Tanaka & Seals, 2008). Many of these athletes want to improve their performance but the ageing process is associated with declines in athletic capability associated with reduced muscle mass (sarcopenia), strength and power (dynapenia; Brunner et al., 2007; Clark & Manini, 2008; Fell & Williams, 2008; Narici, Reeves, Morse & Maganaris, 2004; Welle, 2002). Examples include a ~1% per annum reduction in maximal velocity and power output during 30 m sprinting between the ages of 39 and 96 years (Pantoja, Villareal, Brisswalter, Peyre-Tartaruga & Morin 2016), and a 25% differential in weightlifting

performance (World Records) among those in the 30 and 60-year-old age categories (Baker & Tang, 2010).

## 2.2 Age-associated changes in muscle performance

### *2.2.1 Muscle mass*

Cross-sectional data from 468 men and women shows that between the ages of 18 and 88 years there is a ~27% loss of whole body muscle mass (Janssen, Heymsfield, Wang & Ross, 2000). Similarly, in a 12-year follow-up study, Frontera and colleagues (2000) observed ~12.5% loss of thigh cross-sectional area from the ages of 65 to 77 years in healthy subjects. This loss (e.g. Figure 2.1; atrophy) is caused by a reduction in the size and number of the muscle fibres, in particular type 2 fibres (Lexell, 1995; Lexell, Taylor & Sjostrom, 1988; McPhee et al., 2018; Nilwik et al., 2013). Interestingly, these changes are not uniform in all muscle groups; the age-associated atrophy appears to be site-specific with the lower-limbs undergoing a greater amount than the muscles of the upper-limb (Abe et al., 2011; Abe, Loenneke, Thiebaud & Fukunaga, 2014a; Janssen et al., 2001). Reductions in physical activity with age may account for this specificity (Janssen et al., 2001), especially given that lower-body movements tend to be supported by upper-body contributions with age (e.g. using the body to rise from the chair; Janssen et al., 2001; Macaluso & DeVito, 2004). Additionally, with ageing the lower-body may undergo more severe alterations in connective tissue (e.g. greater increases in fat and connective tissue) than the upper-body (Lynch et al., 1999; Overend et al., 1992), and certain muscle groups (anterior thigh) experience more atrophy than others (posterior thigh). For example, Abe et al. (2011) noted moderate inverse correlations ($r$ = -0.529) between age and quadriceps muscle thickness, yet no relationship between age and hamstring thickness ($r$ = 0.068). Frontera et al. (2008)

also reported a 5.7% decrease in anterior thigh cross-sectional area and non-significant changes (-3.2%) in the posterior thigh muscles after an 8.9 year follow-up from the ages of 71 to 80 years. Abe et al. (2011) suggested declines in participation of high intensity activities with age might contribute to these findings. Importantly, much of the age-associated muscle mass data focuses on those older than 60 years, thus there is a paucity of data regarding the muscle mass changes in middle-aged adults, especially in those who purposefully seek to enhance their musculature via resistance training.

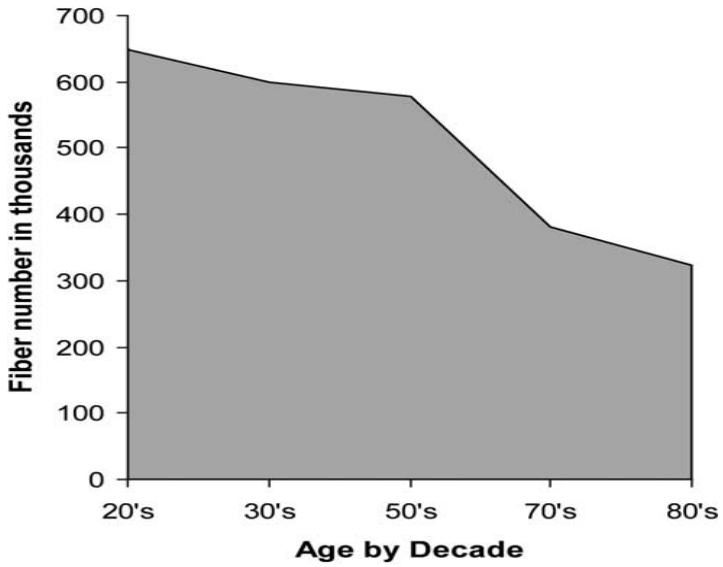


**Figure 2.1** Changes in muscle fibre number in the vastus lateralis with ageing (Greenlund & Nair, 2003, adapted from Lexell et al., 1988).

*2.2.2 Changes in body composition*

Beyond the loss of muscle mass with ageing, old muscle is subject to an infiltration of adipose tissue (also know as intra- and inter-muscular adipose tissue, IMAT; Boettcher et al., 2009; Forsberg et al., 1991; Goodpaster et al., 2001; Jubrias et al., 1997; Zoico et al., 2010). Work by Runge et al. (2004) suggests that this infiltration may result in

relative changes in body composition. That is, body mass is unchanged with age but the composition of the mass is altered. It is also plausible that that these losses in muscle mass with age, and increases in adipose tissue result in a higher body fat percentage in older adults. Notably, increases in IMAT are associated with reduced strength and muscle function (Marcus et al., 2012; Visser et al., 2005) because adipose tissue does not contribute to the body's force generating capacity. Although the mechanisms for IMAT are unclear (Reinders et al., 2016) this increase with age may be partly explained by physical activity levels. For example, Kent-Braun et al. (2000) found a moderate negative correlation between physical activity and noncontracticle tissue (i.e. adipose tissue) in the muscle of older adults. Moreover, IMAT may also develop as a consequence of both a decreased capacity of the subcutaneous tissues to store adipocytes and decreases in fatty acid oxidation (Hamrick, McGee-Lawrence & Frechette, 2016).

### *2.2.3 Muscular strength*

Age-associated strength loss (i.e. dynapenia) has been well researched using isometric and dynamic contractions, with studies often comparing young and older men and women (Anton, Spirduso & Tanaka, 2004; Dalton et al., 2015; Dalton et al., 2012; Doherty, 2003; Frontera, Hughes, Lutz & Evans, 1991). The muscles of the quadriceps, because of their functional importance and accessibility, are the most frequently tested muscle group (Doherty, 2003). Age-related differences in the strength of lower-limb muscles can vary between 20 and 40% when comparing participants in their 60s and 70s to younger (~20s) adults (Doherty, 2003; Larsson, Grimby & Karlsson, 1979; Murray, Gardner, Mollinger & Sepic, 1980).

Losses in strength are present in all muscles but some appear to experience greater decrements. It seems that the lower extremity is more affected than the upper extremity (Candow & Chilibeck, 2005; Frontera et al., 2000; Lynch et al., 1999), though the reasons for such differences are not entirely clear. For example, Nogueira et al. (2013) found among younger (~23 years) and older men (~62 years) that elbow flexor strength was maintained to a greater extent than the muscles of the knee extensor. Macaluso and De Vito (2004) proposed that older people supplement weaker lower-body movements with upper-body ones (as above, using the upper-body to rise from a chair). More mechanistic theories suggest that the lower-body may undergo more severe changes in muscle contractile properties (e.g. decreases in the specific tension of type 1 and 2 fibres; Larsson, Li & Frontera, 1997) and connective tissues (e.g. collagen cross-linking, via advanced glycation end-products, and ensuing tissue stiffness; Haus, Carrithers, Trappe & Trappe, 2007).

### *2.2.4 Muscular power*

Like the losses in muscle mass and strength, age-associated changes in power are well-documented (Byrne, Faure, Keene & Lamb, 2016; Candow & Chilibeck, 2005; Izquierdo et al., 1999; Newton et al., 2002; Petrella, Kim, Tuggle, Hall & Bamman, 2005; Petrella, Kim, Tuggle & Bamman, 2007; Raj, 2010; Runge, Rittweger, Russo, Schiessl & Felsenberg, 2004). For example, during elbow and knee flexion and extensions, older males (~66 years) produced significantly lower power than their younger (~23 years) counterparts at both slow (1.05 $rad \cdot s^{-1}$) and fast (3.14 $rad \cdot s^{-1}$) movement velocities (Candow & Chilibeck, 2005). Arguably, as the groups studied by Candow and Chilibeck (2005) were not physically active, the older group might have been more susceptible to these age-associated decrements in muscle power. Yet,

when physical activity, but not resistance training, was matched between young (~23 years) and old (~62 years) males, these age-associated differences remained (Nogueira et al., 2013). During more complex multi-jointed tasks (i.e. bench throws and countermovement jumps) performed by habitually active males, Izquierdo et al. (1999) also observed an impaired upper- and lower-body power production in older (60 to 74-year-olds) compared to middle-aged (35 to 46-year-olds) adults. When compared to age-associated losses in strength, these losses in power demonstrate faster rates of decline with ageing (Candow & Chilibeck, 2005; Metter, Conwit, Tobin, Forzard, 1997; Petrella et al., 2005) and are likely unfavourable for the middle-aged athlete given the strong relationship between power and sporting tasks (Cronin & Hansen, 2005; Delaney et al., 2015; Lopez-Segovia, Dellal, Chamari & Gonzalez-Badillo, 2014; Loturco et al., 2016).

## 2.3 Mechanisms of sarcopenia and dynapenia

### *2.3.1 Increased physical inactivity*

Ageing is typically associated with reduced overall (Fiatarone & Evans, 1993) and leisure-time physical activity (Combie et al., 2004), despite some ‘veterans’ achieving high activity levels (Tanaka & Seals, 2008; Welle, 2002). Crombie et al. (2004) observed that many elderly people became increasingly inactive owing to physical symptoms (e.g. painful joints, shortness of breath), a reluctance to go out (in the evening) and fears about their physical capabilities (e.g. possibility of falling). Interestingly, the propensity to be less active with ageing is not solely displayed in humans, but occurs as a universal biological phenomenon occurring in all species (Welle, 2002). In humans, reduced physical activity with ageing may be a consequence of time spent involved in other things, such as working (Weir et al., 2002)

and family-related responsibilities (Tanaka & Seals, 2008). Therefore, it appears that sarcopenia and dynapenia make physical activity more difficult, rendering inactivity a consequence of ageing rather than a cause of ageing (Welle, 2002). However, it should be noted that regular exercise can ameliorate the mobility declines associated with sarcopenia (ACSM, 2009).

### *2.3.2 Impaired muscle protein synthesis*

Changes in muscle morphology, for example muscle hypertrophy or repair, are often the consequences of greater muscle protein synthesis (MPS) rather than muscle protein breakdown that results in a positive protein balance (Chesley, MacDougall, Tarnopolsky, Atkinson & Smith, 1992; Folland & Williams, 2007; Schoenfeld, 2010). Conversely, if the muscle is in a state of negative protein balance then muscle wasting will occur (Burd, Gorissen & Van Loon, 2013). Early work using muscle biopsies taken from the vastus lateralis, suggested that negative protein balance may be responsible for sarcopenia with ageing (Balagopla, Rooyackers, Adey, Ades & Nair, 1997; Yarasheski et al., 1999; Welle, Thornton, Jozefowicz & Statt, 1993), whilst more recent studies have not observed differences in protein balance between young (~28 to 34 years) and older (~67 to 70 years) adults (Paddon-Jones et al., 2003; Volpi, Sheffield-Moore, Rasmuseen & Wolfe, 2001). Importantly, a slowed rate of MPS has only been demonstrated in the vastus lateralis of older adults (Balagopla et al., 1997; Yarasheski et al., 1999; Welle et al., 1993) and it remains unknown if changes in MPS occur in other muscle groups of middle-aged adults.

Resistance exercise and the ingestion of amino acids are able to stimulate increases in MPS (Chesley et al., 1992; Folland & Williams, 2007; Koopman, Saris, Wagenmakers & Van Loon, 2007; Kumar et al., 2009). However, ‘anabolic resistance’

(i.e. impaired MPS) is reported in older adults after resistance exercise and amino acid ingestion. For example, after unilateral knee extension exercise across a range of intensities, Kumar et al. (2009) observed increases in MPS in older adults that were to a lesser extent of the younger group (Figure 2.2). The authors suggested that the blunted response was due to a reduced phosphorylation of p70s6K and 4EBP1, both of which are highly related to MPS (Baar & Esser, 1999; Kumar et al., 2009). Indeed, there has been further evidence of 'anabolic resistance' after essential amino acid supplementation (40 g essential amino acids with 5% leucine; Cuthbertson et al., 2004). In this case Cuthbertson and colleagues (2004) purposefully selected physically mobile, active and healthy males without any grossly detectable evidence of sarcopenia. Like Kumar et al. (2009), Cuthbertson et al. (2004) attributed the decreased anabolic sensitivity to changes in the expression and activation of anabolic signalling pathways.

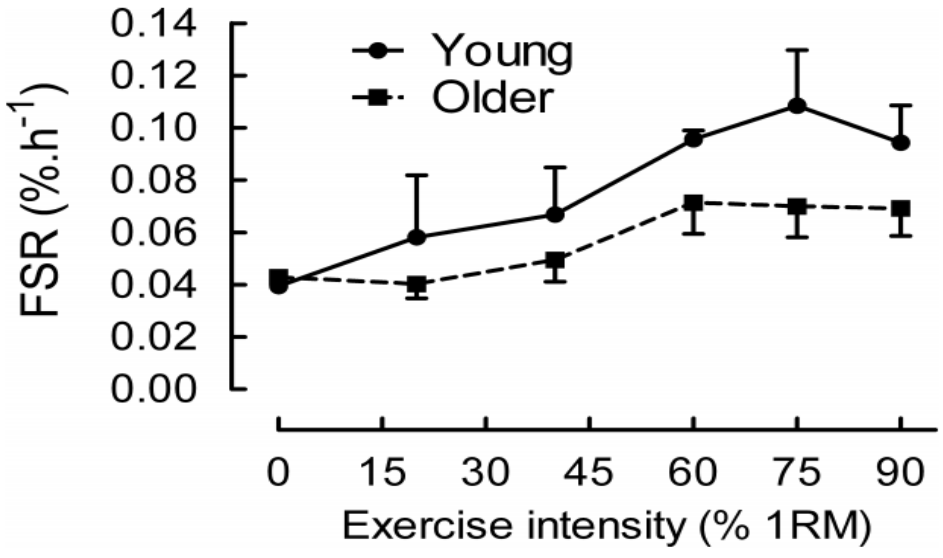


**Figure 2.2** Muscle protein synthesis fractional synthetic rate (% $h^{-1}$) after exercise across a range of intensities measured at 1-2 h post-exercise (Kumar et al., 2008).

*2.3.3 Loss of motor units and remodeling*

Motor units comprise a motor neuron and myofibres that are innervated by a motor unit's axon terminals (McArdle, Katch & Katch, 2009). When a motor unit receives input all of the innervated myofibres contract. However, a motor neuron my innervate multiple myofibres due to the branches of the axon terminal. The maximal force production of a muscle is dependent upon the number of motor units and their firing frequency. With ageing, it has been observed that a decrease in the relative number of motor units occurs in muscles such as the extensor digitorum (Campbell, McComas & Petito, 1973), biceps brachii (Brown et al., 1988 as cited in Welle, 2002), thenar (i.e. thumb abductor; Brown, 1972) and tibialis anterior (de Koning et al., 1988). More recently, such decreases have been reflected in progressively fewer motor units being evident among increasing age groups in the thenar muscles (15-30 years vs. 31-45 years vs. 46-83 years; Yerdelen, Koc & Sarica, 2006). Importantly, this research is the first of its kind to observe a reduced number of motor units specifically in middle-aged adults.

Motor unit remodeling occurs after the death of motor neurons with ageing, leading to denervation of the muscle fibre, atrophy and eventual death (Cartee, Hepple, Bamman & Zierath, 2016; Piasecki et al., 2018; Vella & Kravitz, 2000). Fast twitch motor neurons are preferentially denervated with age and are often reinnervated by adjacent slow twitch motor neurons to offset atrophy (Cartee et al., 2016; Vella & Kravitz, 2000). As slow twitch motor units produce slower, less forceful contractions than fast twitch motor units, the loss of strength and inability to generate high speed movements with ageing is partially explained (Vella & Kravtiz, 2000).

*2.3.4 Reduced proliferation of satellite cells*

Satellite cells are dormant, mononucleated, myogenic cells that reside between the sarcolemma and basal lamina of muscle fibres (Morgan & Partridge, 2003) and regulate pre- and post-natal muscle growth (Zammit et al., 2002). When stimulated externally, these satellite cells are activated and proliferate the injury site to aid the regenerative process (Morgan & Partridge, 2003; Zammit et al., 2002). Welle (2002) propose that to ameliorate sarcopenia, satellite cells must be stimulated through exercise-induced damage to the muscle. This would suggest that to offset losses in muscle mass, the middle-aged male must perform resistance training that damages the muscle.

The precise role of satellite cells in ageing and exercise remains unclear (Pietrangelo et al., 2009). One theory proposes an age-dependent decrease in the satellite cell pool, thus a diminished capacity for repair (Renault, Rolland, Thornell, Mouly & Butler-Browne, 2002). Renault et al. (2002) also observed an accumulation of lipofuscin, which is thought to be responsbile for delayed activation of satellite cells. A recent study in mice documented a blunted hypertrophic response assoicated with a lower satellite cell count, though this did not result in a lower capacity for proliferation (Ballak et al., 2015). This theory, however, was disputed by contrary findings suggesting that satellite cell proportion and morphology are maintained with ageing (Roth et al., 2000). Pietrangelo et al. (2009) proposed that the differences in muscle mass between younger and older individuals may be a product of impaired ability to activate regenerative pathways. Consequently, impaired activation, proliferation and differentiation of satellite cells into myoblasts may lead to age-associated muscle atrophy (Fulle et al., 2005; Pietrangelo et al., (2009). In agreement, Fulle et al. (2005) reported that newborn myoblasts may undergo 55 to 65 divisions while aged (20 year-

old) myoblasts can only undergo 15 to 20 divisions. While it would appear that satellite cell population decreases with age, it does not contribute to the reduced proliferation, activation and differentiation potential.

*2.3.5 Hormonal changes*

Like all cells, myofibres, satellite cells, and motor nerves are regulated by hormones (Welle, 2002). Endocrine signals are produced by organs and transported via the blood to the muscle (Welle, 2002). Testosterone, growth hormone and insulin-like growth factor are commonly referred to as growth factors (or anabolic hormones). To understand the role that hormones play in sarcopenia, their contribution to muscle growth must first be understood. Testosterone principally enhances amino acid uptake and MPS (Kwon & Kravitz, 2004; West & Phillips, 2010) and can activate tissue growth by increasing neurotransmitter presence (Kwon & Kravitz, 2004). Similarly, growth hormone can improve amino acid uptake but additionally induces lipolysis to aid energy provision for muscle growth and connective tissue (Kwon & Kravitz, 2004; West & Phillips, 2010). Finally, insulin-like growth factor aids anabolism by promoting muscle cell repair and enhancing the protein synthetic mechanisms (Kwon & Kravitz, 2004).

Circulating concentrations of these anabolic hormones decline linearly with age (Lamberts, Van den Beld, & Van der Lely, 1997; Perry, 1999), a process refered to as ‘andropause’. Given the importance of these anabolic hormones to muscle growth, it appears that the decreased concentration with ageing helps to explain sarcopenia. However, it should be noted that post-resistance exercise increases in these circulating hormones are not related to hypertrophy, neither acutely nor chronically (over 15 weeks; West et al., 2010). Instead, local mechanisms (i.e. MPS and

phosphorylation of p70S6K), induced by training to failure or with a high volume load (see Section 2.4.2; Burd et al., 2010; Mitchell et al., 2012; Osagawara et al., 2013; Schoenfeld, 2010) are of greater importance to hypertrophy and strength improvements after resistance exercise (West et al., 2010).

### *2.3.6 Oxidative damage*

During cellular metabolism a number of free radicals, such as reactive oxygen species, nitrogen species and reactive aldehydes, are generated (Cartee et al., 2016; Mitchell et al., 2012). With ageing, oxidative stress is increased due to an impaired anti-oxidant buffering and an increase in the free radical production (Mitchell et al., 2012). This can result in damage to muscle components (i.e. myofibrils, mitochondrial proteins, the neuromuscular junction and sarcoplasmic reticulum) which are responsible for muscular contraction (Mitchell et al., 2012). Furthermore, this may contribute to alpha-motor neuron atrophy and the reduced number and function of satellite cells (Cartee et al., 2016; Mitchell et al., 2012). Indeed, ageing has been induced by exposure to free radicals (Dumont et al., 2001; Renault et al., 2002; Von Zglinicki et al., 1995). Although an increase in free radical production has been observed in human models (Capel et al. 2005) in physically active, but not resistance trained, adults there are reports of significant atrophy without increases in mitochondrial reactive oxygen species (Gouspillou et al., 2014). However, apoptic signalling was increased in older men (Gouspillou et al., 2014).

### *2.3.7 Excitation-contraction coupling*

E-C coupling refers to the sequence of events where an action potential is converted to muscle activation and force generation (Miljkovic et al., 2015). In short, electrical

signals are propagated via sarcolemma to the t-tubules. An actional potential causes the release of calcium from the sarcoplasmic reticulum. Subsequently, the calcium ions bind to troponin, which changes shape, thus removing the blocking action of tropomyosin. Theoretically, any impairments to this process with ageing would result in sub-optimal activation of the muscle and a reduction in musc le function (i.e. force or power loss; Clark & Manini, 2012). Such impairments are said to account for the reduction in force-generating capacity with ageing, even when controlling for muscle size (Boncompagni et al., 2006; Petrella et al., 2005).

The influence of age on the calcium release units (i.e. ryanodine receptors) has received the most research attention. For example, Miljkovic and collegues (2015) indicate that the dihyropyridine receptors, which activate calcium release from the sarcoplasmic reticulum via ryanodine receptors, are key to this. Importantly, ageing causes an uncoupling of the dihyropyridine and ryanodine receptors, and an impaired calcium release in rat muscle (Reganathan et al., 1997). Boncompagni and colleagues (2006) reported a reduction in the amount of available release sites for calcium release units (i.e. ryanodine receptors) in aged muscle. The authors suggest that this explains the inefficient calcium release and subsequent impairments in muscular performance that occur with ageing (Boncompagni et al., 2006). Collectively, this data would suggest that impairments in E-C coupling with ageing are a driver for the age-associated losses in strength.

### 2.4 Resistance training and ageing

As the ageing process ultimately manifests itself as decrements in muscle mass, strength and power (see Sections 2.2.1, 2.2.3 and 2.2.4), it is appropriate for those who play sport to involve themselves in activities which might off-set sarcopenia and

dynapenia and help to sustain athletic performance. Undoubtedly, the best method of attaining this is resistance training (Johnston, De Lisio & Parise, 2008; Kosek, Kim, Petrella, Cross & Bamman, 2006; Welle, 2002). Resistance training is a method of physical conditioning which aims to improve a variety of muscle characteristics; usually strength, power and muscle mass. It involves altering repetitions, sets, load and exercises to augment this adaptation.

The earliest studies in this area, which date back to the 1980s, show favourable effects of resistance training on muscular strength and size in older individuals (Frontera, Meredith, O'Reilly, Knuttgen & Evans, 1988; Larsson, 1982). Since this time, there is an increasing number of studies which confirm the former findings. Indeed, these improvements in strength and muscle mass are independent of an athlete's sex or stage in the ageing process (Bottaro, Machado, Nogueira & Veloso, 2007; Kongsgaar, Backer, Jorgensen, Kjaer & Beyer, 2004; Kosek, Kim, Cross & Bamman, 2006; Kraemer et al., 1999; Roth et al., 1999; Roth et al., 2000; Roth et al., 2001a; Roth et al., 2001b; Sayers & Gibson, 2014).

### *2.4.1 Resistance training to offset sarcopenia and dynapenia*

The resistance training (research) interventions typically provided to older populations are of a similar nature to those in young populations. That is, the duration of the intervention can range from four weeks to two years (Sherington & Lord, 1997; McCartney et al., 1996), with weekly frequencies of two to three times (Bottaro et al., 2007; Frontera et al., 1988; Harridge et al., 1999; Hakkinen et al., 2001; Ramirez et al., 2014; Sayers & Gibson, 2010; Sayers & Gibson, 2014). Moreover, exercise intensities range from 40 to 80% of one repetition maximum (1RM; Bottaro et al., 2007; Frontera et al., 1988; Harridge et al., 1999; Hakkinen et al., 2001; Ramirez et al., 2014;

Sayers & Gibson, 2010; Sayers & Gibson, 2014) for ~8 to 15 repetitions (Bottaro et al., 2007; Frontera et al., 1988; Harridge et al., 1999; Hakkinen et al., 2001; Ramirez et al., 2014; Sayers & Gibson, 2010; Sayers & Gibson, 2014) but sometimes as high as 20 to 30 (Larsson, 1992). Researchers will typically alter these variables (i.e. duration, frequency, repetitions and intensity) as well as the methods for assessing strength (e.g. 1RM testing, isokinetic and isometric dynamometry), power and velocity (e.g. force platforms, rotary encoders) and muscle mass changes (e.g. dual x-ray absorptiometry, muscle biopsies). Notwithstanding these methodological variations, post-training changes in strength in older adults range from 25 to 187% (Bottaro et al., 2014; Frontera et al., 1988; Hakkinen et al., 2001; Pyka et al., 1994; Ramirez et al., 2014; Sayers & Gibson, 2010; Taaffe et al., 1999), whilst increases in power are generally lower (4 to 36%; Bottaro et al., 2007; Sayers & Gibson, 2010; Sayers & Gibson, 2014).

Frontera and colleagues (1991) suggested that sarcopenia was a major factor when explaining dynapenia, which implies that resistance training with ageing should focus on hypertrophy, as opposed to strength. However, more recent longitudinal and cross-sectional research has indicated that changes in muscle mass cannot fully account for the changes in strength and power with ageing (Delmonico et al., 2009; Goodpaster et al., 2001; Hughes et al., 2001; Petrella et al., 2005; Visser et al., 2000). Although in young adults, Erskine and colleagues (2014), and more recently Balshaw et al. (2017), found that hypertrophy accounted for only 23 to 28% and 19%, respectively, of the variance in strength gain after a resistance training. Additionally, in 287 participants of ages 19 to 78 years, Ahtiainen et al. (2016) observed a very weak, but significant association between hypertrophy and strength ($r$ = 0.157). Consequently, these data would suggest that hypertrophy has a small contribution to

increases in strength after resistance training. Instead, improvements in strength are said to be due to increases in pennation angle (Erskine et al., 2014) and neural adaptations such as increased central motor drive, reduced pre-synaptic inhibition and increased motor neuron excitability (Aagaard et al., 2002; Balshaw et al., 2017). Additionally, hypertrophy is due to a positive muscle protein balance, driven by an increase in MPS (See Section 2.3.2.; Burd, Mitchell, Churchward-Venne & Phillips, 2012; Burd et al., 2012; West et al., 2009), which results in an expansion of the extracellular matrix and an addition of sarcomeres in series or parallel (Schoenfeld, 2010).

### *2.4.2 Dose-response and resistance training adaptations*

An important consideration when programming resistance training is the dose (i.e. volume or load) given to the participant and the observed response. For example, Radaelli et al. (2015) reported a positive dose-response between the number of sets (1 vs. 3 vs. 5 sets) and hypertrophy over the course of six months. The authors noted that the number of sets was associated with exercise volume (~27,550, ~87,090 and ~161,990 kg for 1, 3 and 5 sets, respectively; Radaelli et al., 2015). These findings have been confirmed in a recent meta-analysis by Schoenfeld, Ogborn and Krieger (2017) who calculated that each weekly set was accompanied by an ES of 0.023 and a muscle gain of 0.37%. The mechanism for this relationship between volume load and hypertrophy appear to be related to increased anabolic signaling and stimulation of MPS (Schoenfeld et al., 2017).

Notably, when resistance training is performed to muscular failure the influence of volume load is negligible (Mitchell et al., 2012; Ogasawara, Loennke, Thiebaud & Abe, 2013). For example, Mitchell and colleagues (2012) observed increases in

muscle size that were not different between groups training to failure for either three sets of 30% 1RM, one set of 80% 1RM or three sets of 80% 1RM. Interestingly, though all groups increased strength, training at 80% 1RM induced a greater strength gain than training at 30% 1RM (no differences between the 80% 1RM groups; Mitchell et al., 2012). The superiority of high load training for increasing strength, and uniformity of low and high loads when performed to failure for hypertrophy, have been observed previously (Ogasawara et al., 2013) and confirmed in a recent meta-analysis (Schoenfeld, Grgic, Ogborn & Krieger, 2017). This finding indicates that a higher volume of exercise is associated with greater gains in hypertrophy, though resistance exercise performed to failure negates the need for a high exercise volume. In addition, strength gains should be achieved via high load training. Though there is a dearth of literature regarding the dose-response in an ageing population, it is plausible that middle-aged athletes would respond in a similar way to their young counterparts.

### 2.5 Monitoring resistance training load

To determine if a training bout is correctly programmed a coach must be able to quantify the load imposed by that training on the athlete (Scott et al., 2016). For example, during endurance training, the load can be monitored using blood lactate concentrations, various HR metrics (Noble, Borg, Jacobs, Ceci & Kaiser, 1983) and oxygen consumption (Foster et al., 1995), due to their strong association with exercise intensity (Seiler, 2010). However, because of the numerous variables involved when programming resistance training (e.g. rest time, repetitions, volume load; Schoenfeld, 2010), this mode of exercise cannot be easily or consistently quantified using the above measurements (McGuigan & Foster, 2004; Scott et al., 2016; Sweet, Foster, McGuigan & Brice, 2004). Instead, resistance training can be quantified in terms of its

internal and external load*. External* load (i.e. what the athlete does) refers to the work done and, in the context of resistance training, is typically measured in terms of volume load, power output or velocity of movement. The *internal* load refers to the relative physiological or psychological stress on the athlete (i.e. how the athlete responds) and can be influenced by individual physical qualities such as training status (Pierce et al., 1993; Stone et al., 1987), individual strength (John et al., 2009) and age (Smolander et al., 1998).

### *2.5.1 Internal load*

#### *2.5.1.1 Heart rate*

Monitoring HR is a common method of assessing internal load in athletes (Halson, 2014). During endurance and intermittent running protocols HR is typically expressed as percentage of an athlete's peak HR. It is suggested, however, that HR is a poor method of evaluating high-intensity exercise, such as resistance training (Foster et al., 2001), potentially owing to the inherent short exercise bouts and long recovery periods. Nevertheless, several studies have monitored HR responses during resistance training (Kawano, Onodera, Higuuchi & Miyachi, 2008; Pierce, Rozenek & Stone, 1993; Smolander et al. 1998; Suminski et al., 1997). The early research by Stone et al. (1987) observed that when squatting to failure, trained males recorded higher HRs than untrained males, which was associated with a high volume of exercise at muscular failure. Alterations in HR during exercise are a consequence of alterations to sympathetic and parasympathetic activity which seek to increase and decrease HR, respectively (McArdle et al. 2009). Elevations in HR after resistance exercise are said to be associated with cardiac sympathetic activation and parasympathetic deactivation (Rezk, Marrache, Tinucci, Milon Jr, & Forjaz, 2006).

Thus, a higher HR during exercise might be driven by alterations in cardiac sympathetic and parasympathetic activity (Rezk et al., 2006), which aim to increase oxygen delivery to the working musculature.

To the author's knowledge only two studies have determined the HR response to resistance exercise between age groups. Smolander et al. (1998) compared mean absolute HRs during forearm and quadriceps isometric exercise at 20, 40 and 50% maximal voluntary contraction (MVC) in young (~26 years) and older (~57 years) males. The younger males produced higher HRs compared to the older males and the magnitude of these differences was greater at the higher workloads (Smolander et al., 1998). Such differences were attributed to an attenuated vagal tone in the older males (Smolander et al., 1998). During leg press exercise, Kawano et al. (2008) observed no differences in peak HR at relative or absolute exercise intensities in young (~21 years) and middle-aged (~48 years) males. Though the authors did not provide a reason for their findings, it is plausible that the age-associated changes in vagal tone suggested by Smoldander et al. (1998) were not sufficiently different between the two age groups used by Kawano et al. (2008).

*2.5.1.2 Blood lactate concentration*

Typical resistance training involves a high rate of energy usage accompanied by the breakdown of phosphagens, glycogen and glucose (Stone et al., 1987), leading to symptoms of metabolic stress that manifests as lactate accumulation (Lang et al., 1998). This accumulation may increase cell swelling (Lang et al., 1998), a mechanism that has been attributed to facilitate hypertrophy (Schoenfeld, 2010). Consequently, blood lactate concentration response to resistance training is well documented in the literature (Date, 2009; Hakkinen et al., 1998; Manini et al., 2012; Mulligan et al., 1996;

Pierce et al., 1993; Pyka et al., 1997; Smilios, Pilandis, Karamouzis, Parlavantzas & Tokmakidis, 2007; Stone et al., 1987; Suminski et al., 1997; Walker et al., 2013). As expected, protocols with higher volumes (Date, 2009; Mulligan et al., 1996) and those performed at higher intensities (Stone et al., 1987; Suminski et al., 1997) elicit the greatest increases in blood lactate concentration. Furthermore, those who are habitually resistance trained produce higher blood lactate concentrations than their untrained counterparts at the point of muscular failure, possibly owing to an enhanced recruitment of type 2 fibres in trained muscle (Stone et al., 1987).

Several studies have reported greater increases in blood lactate in young (~23 to 28 years) compared to older (~65 to 72 years) males after both upper- and lower-body resistance exercise (Pyka et al., 1997; Smilios et al., 2007; Walker et al., 2013). These findings are attributed to the higher muscle mass and greater proportion of type 2 fibres in young compared to older males (Pyka et al., 1997; Smilios et al., 2007; Walker et al., 2013). Interestingly, at the point of muscular failure, blood lactate increased to a similar extent in young (~28 years) and old (~67 years) males (Manini et al., 2012). While Manini et al. (2012) did not explain these observations, it may be plausible that the point of muscular failure is associated with a similar reliance on glycolytic pathways and/or a lactate production and clearance in these young and old groups.

*2.5.1.3 Ratings of perceived exertion*

RPE refers to an individual's perception of exertion during physical activity (Borg, 1982) and can be defined as "the conscious sensation of how hard, heavy and strenuous a physical task is" (Marcora, 2010, p. 455). RPE is a common method of assessing internal load that requires an athlete to monitor their perception of the

physiological intensity with an alpha-numeric scale during exercise, or retrospectively a short time after exercise (Halson, 2014). It is suggested that perception of exertion (or effort) reflects central motor command to the muscles (deMorree, Klein & Marcora, 2012; Marcora, 2009), though some physiologists believe that fatigue-related metabolites stimulate sensory afferents to generate perception of effort (Amann et al., 2010; St. Clair Gibson et al., 2006). The interested reader is directed to the excellent reviews of St. Clair Gibson et al. (2006) and Pereira et al. (2014).

Notwithstanding these mechanisms, resistance exercise loads have been monitored with the traditional 15-point Borg scale (Gearhart, Lagally, Jakicic, Gallagher, Robertson, 2002; Lagally, et al., 2002), the modified CR-10 scale (John et al., 2004; Sweet, Foster, McGuigan & Brice, 2004) and the OMNI-RPE scale (Gearhart, Lagally, Riechman, Andrews & Robertson, 2009; Robertson et al., 2003). The OMNI-RPE scale is a 10-point scale like the CR-10, but contains additional verbal and pictorial anchors related to resistance training. Gearhart et al. (2002) and Suminski et al. (1997) noted that athletes could use RPE to detect differences in the exercise intensity, whilst Latif (2008) observed higher RPEs reported with greater exercise volumes. After strength increases from resistance training, perceived exertion for a given absolute load is lowered (Pierce, Rozenek & Stone, 1993). Collectively, these data suggest the RPE measures are sensitive to changes in exercise intensity and volume load.

Regarding the age-related differences in RPE, Manini et al. (2012) reported no differences in RPE after knee extension exercise to failure between young (~28 years) and old (~67 years) males, despite the young group completing a greater exercise volume. Conversely, Allman and Rice (2003) noted higher CR-10 RPE, but similar time to fatigue, in older (~84 years) men when compared to their young counterparts

(~25 years) during the early stages of fatiguing isometric elbow flexion exercise. However, at the point of fatigue both groups reported similar RPEs. These observations might be supported to some extent by the work of John and colleagues (2009), who had young (~29 years) and older (~71 years) participants perform elbow flexion and extension exercise at prescribed CR-10 RPE levels (1, 3, 5, 7, and 9). From RPEs of 3 to 9, the young group produced a greater absolute and relative force than the old group, which the authors attributed to the older participants needing to increase their motor command, and subsequently the magnitude of force, to match the effort (John et al., 2009). Of note however, is that the use of RPE in this way (in its 'production mode') involves different cognitive and feedback processes to that of passively responding to a request of 'how hard does it feel now' (when RPE is used in its 'estimation mode'). In general, the influence of age on RPE responses during resistance training is unclear.

*2.5.1.4 Session ratings of perceived exertion*

Session RPE (sRPE) requires an individual to retrospectively (~15 to 30 minutes post-exercise) provide a global indication of the intensity of the session based on the CR-10 scale (McGuigan & Foster, 2004). Though typically used to quantify the internal load for steady-state and intermittent (Foster et al., 2015) exercise, this method has been deemed a valid (Sweet et al., 2004) and reliable (Day, McGuigan, Brice & Foster, 2004; McGuigan, Egan & Foster, 2004) indicator of resistance exercise intensity. A further benefit of this measure is that athletes are only required to provide a single rating after exercise (Scott et al., 2016). Indeed, Kraft, Green and Thompson (2014) reported no difference in sRPE measured at 15 and 30 minutes after resistance exercise. It has been proposed that sRPE is able to detect differences in exercise

intensity (Day et al., 2004; McGuigan et al., 2004), though it may provide an underestimation when compared to RPE taken immediately post-exercise (Sweet et al., 2004). Additionally, Genner and Weston (2014) found that sRPE was better related ($r$ = .55) to volume load than exercise intensity, a finding supported by Pritchett, Green Wickwire, Pritchett and Kovacs (2009). To the author's knowledge, no study has sought to compare the sRPE scores in young and middle-aged males.

### *2.5.2 External load*

#### *2.5.2.1 Volume load*

The absolute volume load (sets x repetitions x load) is an extension of the repetition method (i.e. the total number of repetitions completed) but provides a more robust method to monitor training load (Kraemer & Ratamess, 2004; Scott et al., 2016). While the method is attractive due to its simplicity and strong relationship to internal load measures (Genner & Weston, 2014), it is not without limitations (McGuigan & Foster, 2004; Scott et al., 2016). Volume load does not account for rest periods between sets and the velocity of the repetitions. Training status, fatigue and, potentially, age might also influence the efficacy of the volume load method to quantify training load (McGuigan & Foster, 2004). Furthermore, this method cannot be used to compare the load between individuals as it does not account for the relative intensity (e.g. %1RM) each athlete is using (Scott et al., 2016). For example, if athlete A has a greater 1RM than athlete B, and both perform three sets of 10 repetitions, the absolute load for athlete A will be higher, despite both working at the same relative intensity. Thus, calculating the relative volume load (sets x repetitions x %1RM) may account for this (Scott et al., 2016).

Whilst relative volume provides a good reflection of the training stimulus, it does not account for the repetitions performed in each set, that is, the more repetitions performed in a set, the higher the intensity. For example, three sets of 10 repetitions at 70% 1RM will be more demanding than 10 sets of three repetitions at 70% 1RM, despite the absolute and relative load being identical. It is possible to calculate each athlete's repetition maximum load using prediction equations to equate the volume load to a specific repetition maximum (Scott et al., 2016). However, if rest periods are altered during training then the repetition maximum for each is not accounted for (Scott et al., (2016). Regardless of the method of calculating volume load, the demands imposed on the athlete can be misrepresented (McGuigan & Foster, 2004; Scott et al., 2016).

*2.5.2.2 Repetition velocity and power*

Muscle functional variables are routinely measured using MVC under isometric (i.e. against a fixed arm), isokinetic (i.e. at a constant angular velocity) or isotonic (i.e. a change in muscle length but not tension) conditions. These approaches have been used to assess velocity and power during exercise (Dalton et al., 2012; Dalton et al., 2015), the magnitude of muscle-damage (Burt et al., 2014; Burt & Twist, 2011; Byrne et al., 2001; Davies et al., 2008; Davies et al., 2011; Doncaster & Twist, 2012), adaptations to training (Burgomaster et al., 2003; Young & Bilby, 1993) and differences between populations of interest (Aoki & Demura, 2011; Candow & Chilibeck, 2005; Frontera et al., 2000; Petrella et al., 2007). However, the single-jointed nature of these movements fail to replicate the multi-jointed and dynamic characteristics of sporting movements and resistance training (Flavo & Bloomer, 2006). Moreover, the non-involvement of the stretch-shortening cycle during isometric

actions (Marcora & Miller, 2000) and the poor to moderate relationship with measures of athletic performance (Marcora & Miller, 2000; Young & Bilby, 1993) puts the ecological validity of such tools into question.

The emergence of portable technologies (e.g. rotary encoders) enables the practitioner to measure a wide range of external load metrics during multi-jointed resistance exercise (Scott et al., 2016). Rotary encoders attach to a barbell to record displacement and thus velocity, from which acceleration and power can be derived (Harris, Cronin, Taylor, Borris & Sheppard, 2010). A distinct benefit of these methods is that they can provide immediate feedback on training load to the coach and athlete between repetitions (Scott et al., 2016). Recording these metrics can have important implications for monitoring resistance training, both acutely and chronically (Scott et al., 2016).

Importantly, linear position transducers provide an objective method of monitoring the repetition velocity and power, during resistance exercise. For example, Gonzalez-Badillo and Sanchez-Medina (2010) determined that monitoring mean velocity during the first repetition of a set made it possible to determine the %1RM that was being used because of the stability of the load-velocity relationship. Moreover, each increase in 5% 1RM resulted in 0.07 to 0.09 $m \cdot s^{-1}$ change in velocity during bench press exercise (Gonzalez-Badillo & Sanchez-Medina, 2010). Thus, if an athlete improves their velocity by 0.07 to 0.09 $m \cdot s^{-1}$ 1RM would have improved by 5%, making it possible to monitor training load and adaptations using velocity (Gonzalez-Badillo & Sanchez-Medina, 2010).

A further application of velocity measurement is to determine fatigue. Research by Sanchez-Medina and Gonzalez-Badillo (2011) reported that measuring repetition velocity (i.e. the loss of velocity) provided a good estimation of neuromuscular fatigue

and metabolic stress during 15 resistance bench press and squat exercise protocols. When comparing velocity loss, by converting angular velocity to linear velocity) in young (~27 years) and old (~64 years) males and females during 10 repetitions of knee extension exercise at the 40% MVC load, the older group displayed a greater rate of velocity loss (Petrella et al., 2005). However, for power during 10 repetitions of sit-to-stand exercise there were no differences in the rate of power loss (Petrella et al., 2005). Petrella et al. (2005) suggest that the difference in fatigue rates for knee extension and sit-to-stand exercise was due to task specificity. That is, both groups would typically perform sit-to-stand exercise, but the older group would not be accustomed to knee extensions. Moreover, older men (~75 years) experience greater losses in power during dynamic (isotonic), but not isokinetic, knee extension exercise to failure than their young counterparts (~25 years; Dalton et al., 2015). There exists a paucity of data in this area and future studies should seek to assess the fatigue profiles of middle-aged males, especially during multi-jointed dynamic exercise that would usually be incorporated into their resistance training programmes.

### *2.5.3 Considerations in the assessment of velocity and power*

Before measurement tools can be incorporated into research, their reliability should be determined. That is, the measurement error that these tools produce must be known, otherwise changes in muscle function may be masked by measurement error.

#### *2.5.3.1 Reliability of rotary encoders*

Reliability refers to the reproducibility of a given tool, that is, the degree to which a tool produces the same scores when applied in the same circumstances. Thus, a reliability study is required to determine the test-retest reliability of a measure (Batterham &

George, 2003). Batterham and George (2003) indicate that reliability is a prerequisite for validity because a measurement tool cannot be a valid test if it does not produce repeatable results. Consequently, before a measurement tool is incorporated into research its reliability must be determined. Jennings and colleagues (2005) attempted to determine the inter-day reliability of the FitroDyne rotary encoder for the assessment of peak power during single-jointed (bicep curl) and ballistic (squat jump) exercise. The authors noted strong intra-class correlations ($r$ = 0.97 for both exercises) and limits of agreement of -17 ± 96.0 W and 0.11 ± 13.6 for squat jump and bicep curl, respectively. However, applied practitioners may prescribe loads to athletes that do not produce peak power (i.e. during low-force, high-velocity or high-force, low-velocity training). As such, Stock et al. (2011) sought to determine the inter-reliability of peak velocity using the FitroDyne at loads corresponding to 10 to 90% 1RM. From loads of 10 to 70% 1RM, the authors observed moderate to strong intraclass correlation coefficients ($r$ = 0.57 to 0.81) and load coefficients of variation (CV; 3.1 to 5.8%). However, at 80 and 90% 1RM, CVs of 10.3 and 12.6%, respectively, indicated poor reliability.

*2.5.3.2 Validity of rotary encoders*

Only Garnacho-Castano and colleagues (2015) have sought to determine the concurrent validity of the FitroDyne, albeit they failed to establish its criterion validity by comparing to a ‘gold standard’ method (i.e. video analysis or a force platform). Garnacho-Castano et al. (2015) reported strong intra-class correlation coefficients ( > 0.9) for peak and mean velocity between the FitroDyne and the T-Force Dynamic Measurement System during back squat and bench press exercises. However, the authors also failed to generate any measures of absolute validity and deliberated on

the practical significance of the within-participant differences (errors) observed. Indeed, recent work has noted that such methods (correlations and null hypothesis tests) are inappropriate for measuring agreement (McLaughlin, 2013; Mundy et al., 2016). There have, however, been two studies which have established the validity of a rotary encoder against criterion methods. For example, Crewther et al. (2011) compared peak power assessed using a rotary encoder and force plate during squat jumps and reported moderate to strong correlations ($r$ = 0.62 to 0.82) accompanied by large random errors (± 611 to 879 W) at loads of 20, 40, 60 and 80 kg. It should be noted that rotary encoders measure barbell velocity, whilst force plates record ground reaction forces. These differences in the metrics being recorded might account for the poor agreement. Indeed, when the rotary encoders were compared to video analysis during bench press and squat exercise, Drinkwater et al. (2007) observed strong correlations ($r$ = 0.97 to 1.00) and small CVs (CV% = 1.08 to 3.01).

### 2.6 Acute responses to resistance exercise

While the use of the term ‘damage’ to reflect alterations to muscle after eccentric contractions has been debated (Hyldahl & Hubal, 2014), a consequence of resistance exercise, particularly when it is unaccustomed and comprising high volume, is damage to the muscle ultrastructure (i.e. Z-line streaming). Associated symptoms are also often present, including delayed onset muscle soreness, increases in specific intramuscular proteins (e.g. muscle-specific creatine kinase (CK), myoglobin), swelling of the affected limb, decreased range of motion and impaired muscle force producing capacity (Byrne, Twist & Eston, 2004; Damas et al., 2016; Hyldahl & Hubal, 2014).

It is well documented that eccentric muscle actions produce the greatest amount of force (Tsesh, Dudley, Duvoisin, Hather & Harris, 1990; Westing, Cresswell

& Thorstensson, 1991) and invoke the most severe muscle damage (Bryne et al., 2004; Eston et al., 2003). While concentric force production occurs via the hydrolysis of adenosine triphosphate (ATP) to attached and detach actin-myosin cross bridges, eccentric contractions are not ATP-dependant (McHugh, Connolly, Eston & Gleim, 1999). During eccentric muscle actions, the muscle is forcibly lengthened, via external force, whilst under tension (Clarkson & Sayers, 1999; Eston et al., 2003). This places a high strain on the muscle ultrastructure because fewer motor units are activated than in concentric contractions (Enoka, 1996; Kellis & Baltzopoulos, 1998; McHugh, Connolly, Eston & Gleim, 2000). The magnitude of damage resulting from eccentric actions is exacerbated when performed at longer muscle lengths (Child, Saxton & Donnelly, 1998; Nosaka & Sakamoto, 2001), with greater forces (Nosaka, Newton, & Sacco, 2002; Nosaka & Sakamoto, 2001) and at faster angular velocities (Chapman, Newton, Sacco, & Nosaka, 2006). The susceptibility of a muscle to damage might also be reduced depending on its previous exposure to eccentric exercise (Paulsen et al., 2009; Stupka, Tarnopolsky, Yardley, & Phillips, 2001). This protective adaptation is known as the repeated bout effect (RBE; McHugh, 2003; Hyldahl, Chen & Nosaka, 2017). Differences in habitual loading of limbs could also explain the increased susceptibility to damaging exercise for upper compared to lower-body muscle groups (Chen, Lin, Chen, Lin & Nosaka, 2011).

### *2.6.1 Proposed mechanisms initiating EIMD*

While several studies have attempted to describe the events that follow muscle damaging exercise (e.g. Armstrong et al., 1994; Armstrong, 1990; Prose & Morgan, 2001; Kendall & Eston, 2002; Hyldahl et al., 2014; Peake et al., 2017), this complex process can be simplified into two phases: (i) the initial phase or primary damage that

occurs after the mechanical work performed; and (ii) secondary damage that proliferates tissue damage through processes associated with the inflammatory response.

*2.6.1.1 Primary muscle damage*

Although several metabolic factors (i.e. insufficient mitochondrial respiration, lowered pH and increased muscular temperature) have been proposed as mechanisms of primary damage during eccentric exercise (Armstrong, 1990; Armstrong, Warren & Waren, 1991; Bigland-Ritchie & Woods; 1976; Robergs et al., 1991; Tee et al., 2007), mechanical loading of muscle during exercise is the most likely candidiate (Friden & Lieber, 1992; Proske & Morgan, 2001). It is proposed that structural components (e.g. sarcolemma and sarcomere) of a muscle fibre rupture if the tensile stress on the fibre is greater than its tensile strength (Armstrong et al., 1991). This has been attributed to an increase stress per muscle fibre that is consistent with eccentric contractions (Enoka, 1996). This altered loading profile, i.e. high force with low fibre recruitment, places a strain on the myofibres and when combined with repeated muscular contractions results in damage to the myofibres (Jones, Newham & Torgan, 1989).

*2.6.1.2 The 'popping sarcomere' hypothesis*

The 'popping sarcomere' hypothesis proposes EIMD occurs on the descending limb of the length-tension curve (Figure 2.3 Proske & Morgan, 2001). During eccentric contractions myofibrils are stretched whilst contracting, and some sarcomeres resist the stretch more than others (Morgan, 1990; Proske & Allen, 2005). During a series of eccentric actions, this mechanism results in, for the most part, non-uniform lengthening of weaker sarcomeres (Byrne et al., 2004; Morgan, 1990; Morgan & Allen,

1999; Proske & Morgan 2001). The sarcomeres become progressively weaker until they reach a point of uncontrollable lengthening characterised by an absence of myofilament overlapping and inability to re-interdigitate (Byrne et al., 2004; Morgan, 1990; Morgan & Allen, 1999; Proske & Morgan 2001). When the myofibre relaxes, the lengthened sarcomeres remain overextended which is illustrated by a rightward shift in the length-tension relationship (Morgan, 1990; Morgan & Allen, 1999; Proske & Morgan, 2001).

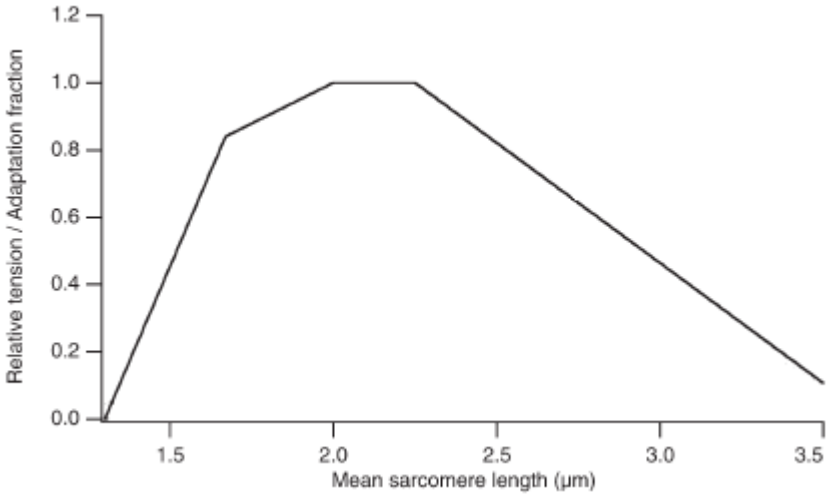


**Figure 2.3** The length-tension relationship (Proske & Morgan, 2001).

Proske and Morgan (2001) further suggest that excessive lengthening of the muscle disrupts the passive tissues (i.e. connective tissue) which provide support to the sarcomeres. Friden and Leiber (2001) indicate that this also causes Z-lines to appear wavy (Figure 2.4). In extreme cases, the Z-lines widen and displace material into the neighbouring sarcomeres (Friden & Leiber, 2001). The damaged muscle fibres show a shift in optimum length for tension in the way of longer muscle lengths (Byrne et al., 2004; Proske & Morgan, 2001). These aforementioned alterations are commonly

thought to contribute to force decrements (Damas et al., 2016; McCully & Faulkner, 1985).

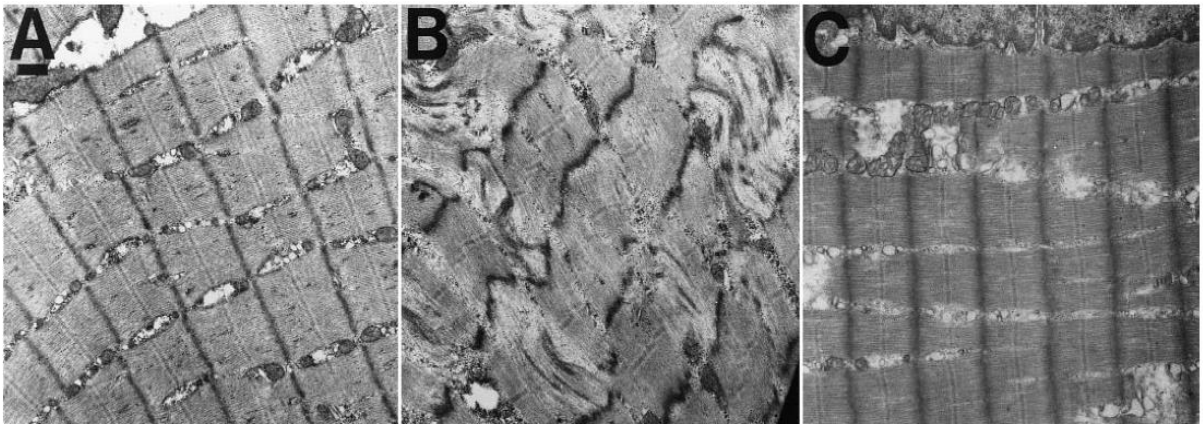


**Figure 2.4** Human vastus lateralis before (A), 2 (B) and 7 days (C) after 10 x 10 eccentric muscle actions illustrates Z-line streaming (Hortobagyi et al., 1998).

*2.6.1.3 Excitation-contraction coupling failure*

In undamaged muscle fibres, an action potential triggers a depolarisation of the transverse tubular system which stimulates a rapid influx of calcium from the sarcoplasmic reticulum (SR) through the lateral sacs of the terminal cisternae (Kim & Vergara, 1998; McArdle et al., 2009). The binding of calcium to troponin C on the actin filaments acts to uncover the cross-bridge binding sites allowing the action of contractile proteins and ultimately resulting in muscular contraction (McArdle et al., 2009). Once this action potential ceases, calcium is actively transported back into the sarcoplasmic reticulum (McArdle et al., 2009). This is called the E-C coupling process. In damaged muscle tissue, calcium homeostasis is lost due to damage to the t-tubules, sarcoplasmic reticulum and junctophilins, which mediate the complex beween the t-tubules and sarcoplasmic reticulum (Byrne et al., 2004; Corona et al., 2010; Proske & Morgan, 2001; Morgan & Allen, 1999). The rate of calcium release is reduced and

thought to be localised to damaged regions (Byrne et al., 2004; Morgan & Allen, 1999). The principal factor contributing to force decrements, at both maximal and sub-maximal intensities, is the resultant failure to fully stimulate contractile machinery rather than damage to them (Byrne et al., 2004). Indeed, E-C coupling failure is demonstrated via the force-frequency relationship (Figure 2.5; Hill et al., 2001). When a muscle is stimulated through a range of frequencies, it exhibits greater force losses at low frequencies than high frequencies (Byrne et al., 2004; Eston et al., 2003; Edwards, Hill, Jones & Merton, 1977; Hill et al., 2001). This phenomenon is termed low frequency fatigue.

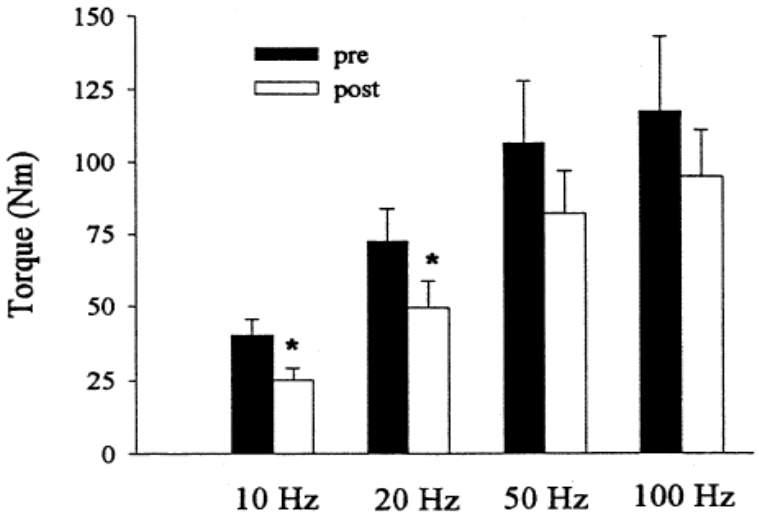


**Figure 2.5** Force-frequency relationship post-EIMD from leg extension exercise. *denotes significantly different from pre. Figure from Hill et al. (2001).

Early literature suggests less calcium release per action potential (Edwards et al., 1977; Figure 2.8) and this has since been confirmed elsewhere (Ingalls, Warren, Williams, Ward & Armstrong, 1998; Warren et al., 1993). Allen (2001) however, suggests that the increased compliance of weakened sarcomeres will inhibit force

production at low frequencies. Therefore, reductions of greater force decrements at low frequencies may might also reflect inceased sarcomere compliance, not just E-C coupling failure (Allen, 2001; Morgan & Allen, 2007). Moreover, E-C coupling failure does not wholly explain the rightward shift in the length-tension relationship (Allen, 2001; Byrne et al., 2004; Eston et al., 2003; Proske & Allen, 2005). Consequently, it appears that E-C coupling failure is secondary to mechanical damage to the myofibre (Allen, 2001; Proske & Allen, 2005). This review of literature supports the popular series of events put forward by Proske and Allen (2001; Figure 2.6).

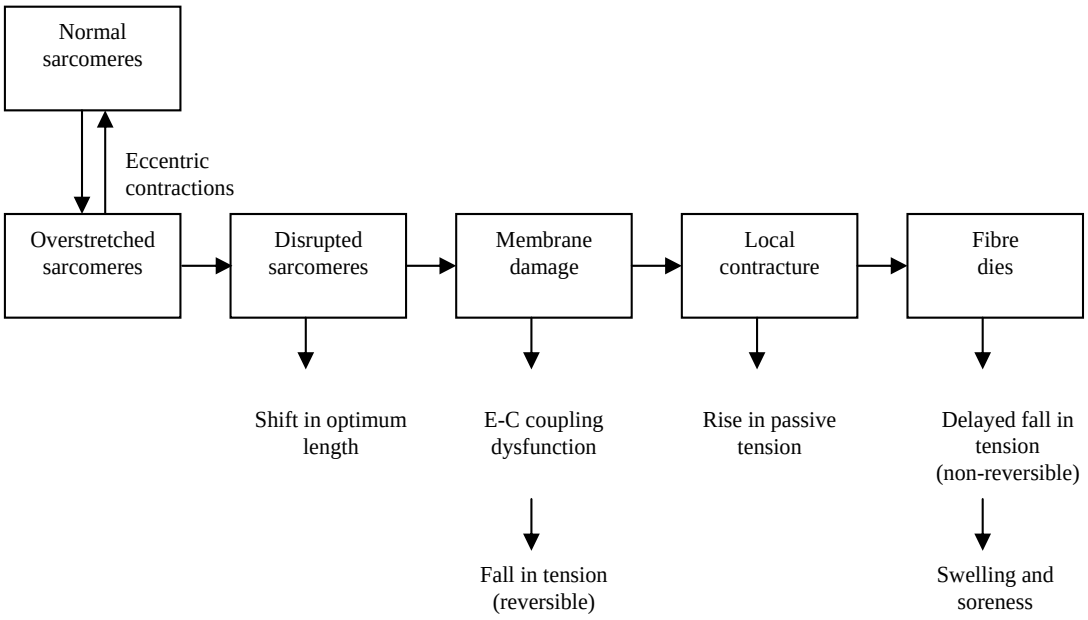


**Figure 2.6** Postulated series of events leadings to muscle damage from eccentric exercise (Proske & Morgan, 2001; p. 334).

*2.6.2 Secondary muscle damage*

After the initial event there is a failing in the structure of the myofibres (Armstrong et al., 1991). Subsequently calcium, enters the cytoplasm in an uncontrolled manner, thus triggering the autogenic events (Armstrong et al., 1991; Kendall & Eston, 2002).

Indeed, calcium plays a vital role in muscular contraction via its attachment of actin and myosin (McArdle et al., 2009). When the cytoplasm holds high concentrations of calcium, a number of harmful events occur to exacerbate damage (Armstrong, 1984; Ebbeling & Clarkson, 1989; Gissel, 2000; Gissel, 2005; Howartson & Van Someren, 2008). Calcium-dependent calpains and phospholipases are said to be activated upon detection of high intracellular calcium concentrations (Armstrong et al., 1991; Gissel, 2005; Howartson & Van Someren, 2008). While the mitochondria can store large concentrations of calcium, any excess leads to the production of reactive oxygen species which damage the membrane (Gissel, 2005; Kendall & Eston, 2002). Moreover, excess mitochondrial calcium could produce ischaemic conditions within the myofibre and inhibit ATP production, thereby impairing muscular contraction (Armstrong, 1984; Ebbeling & Clarkson, 1989; Gissel, 2005). Given the role of calcium in stimulating the contractile machinery, excessive realise of calcium might explain the increase in passive tension (e.g. injury contracture) after damaging exercise (Proske & Allen, 2005; Proske & Morgan, 2001). Essentially, the autogenic events result in a loss of calcium homeostasis within the muscle.

#### *2.6.2.1 Phagocytic events in EIMD*

The phagocytic response is present from 1 to 6 h after the initial events and might last 2 to 4 days (Armstrong, 1990; Armstrong et al., 1991; Tidbull, 2005). This mechanism is characterised by an inflammatory response and the removal of damaged tissue and debris, thus allowing regeneration of the myofibres (Armstrong, 1990; Armstrong et al., 1991; Tee et al., 2007). Damaged myofibres that are close to functioning blood vessels become infiltrated with phagocytes (e.g. leukocytes such as neutrophils and

macrophages; Carlson, 1973; Clarkson & Sayers, 1999; Kendall & Eston, 2002; Hikida, Staron, Hagerman, Sherman & Costill, 1983).

Despite differences in opinions it would appear that neutrophils are first to the injury site (Peake et al., 2005; Tidbull, 1995; Tidbull, 2005). The invasion of neutrophils is said to be stimulated by the activation of proteases, such as calpain, after disruption of calcium homeostasis (Belcastro, Shewchuk & Raj, 1998; Clarkson & Sayers, 1999; Gissel, 2005; Kendall & Eston, 2002). Indeed, Raj, Booker and Belcastro (1998) observed that calcium-stimulated proteolysis resulted in neutrophil accumulation within the striated muscle in rats. Furthermore, the authors suggest that calpain was responsible for localising the neutrophil response (Kendall & Eston, 2002; Raj et al., 1998), splitting of protein substrates, including myofibrillar proteins troponin, tropomyosin and myosin light chain, and activation of an adaptive response (Belcastro et al., 1998; Saido, Sorimachi & Suzuki, 1994). It is suggested that neutrophils are unable to differentiate between foreign and host antigens and subsequently damage healthy tissues which surround the site (Clarkson & Sayers, 1999; Kendall & Eston, 2002; MacIntyre et al., 1995; St Pierre Schneider & Tiidus, 2007; Tidbull, 2005).

Another suggested process is that elevated calcium can signal for pro-inflammatory cytokines (Peake et al., 2005) such as tumour necrosis factor or interleukins. Cytokines are polypeptides that activate on the surface of target cells in order to alter cellular function (Kendall & Eston, 2002; MacIntyre et al., 1995). These cytokines are thought to increase leukocyte adhesion and increase macrophage activation (MacIntyre et al., 1995), cause cell necrosis and apoptosis (Chu, 2013). Thereafter, macrophages, which also produce reactive oxygen species, enter the cell which remove cellular debris through phagocytosis (MacIntyre et al., 1995; Tidbull, 1995; Tidbull, 2005). This inflammatory response induces localised cell swelling as a

consequence of oedema (increased fluid and plasma proteins; Clarkson & Sayers, 1999; Kendall & Eston, 2002; Proske & Morgan, 2001). Peak swelling may not occur until 5 to 10 days post as cellular fragments are removed (Clarkson & Sayers, 1999).

#### *2.6.2.2 Regenerative events of EIMD*

The regenerative stage involves the repair of damaged myofibres (Armstrong, 1990). Satellite cells are myogenic precursors to myofibres that reside on the sarcolemma (Welle, 2002). As myofibres are unable to undergo mitosis (cell division) they require satellite cells to regenerate. When activated, satellite cells come out of their dormant state and proliferate on the injury site (Welle, 2002). It is not yet understood whether the phagocytic events are required for satellite cell stimulation (Carlson, 1973; Kendall & Eston, 2002). It would appear, however, that macrophage invasion is necessary to stimulate satellite cell proliferation (Jones & Round, 1990; Kendall & Eston, 2002).

## 2.7 Symptoms of EIMD

EIMD is characterised by symptoms that present both immediately and for up to 14 days after the initial exercise bout. The extent of muscle damage is typically assessed by measuring various indirect markers, which have been reviewed in considerable detail elsewhere (Byrne et al., 2004; Damas et al., 2015; Eston et al., 2003; Hyldahl & Hubal, 2014; Kendall & Eston, 2002; Toft et al., 2002). Accordingly, this section of the review of literature will summarise the key changes in indirect markers of EIMD and then focus on the different responses of these markers between age groups.

### *2.7.1 Muscular strength*

Reduced muscle force after eccentric exercise is considered the most appropriate

indirect marker of muscle damage (Damas et al. 2016; Paulsen, Mikkelsen, Raastad & Peake, 2012; Warren, Lowe & Armstrong, 1999). Depending on the type, intensity and duration of exercise performed, losses in force after exercise are between 15-60% of pre-damage values and can persist for ~2 weeks (Hyldahl, Olson, Welling, Groscost, & Parcell, 2014; Paulsen et al., 2012). The underlying mechanisms are complex and caused by physical damage to the sarcomere and sarcolemma from eccentric lengthening and E-C coupling failure (see section 2.6.1.3). Moreover, a reduction in voluntary activation (VA) might imply impairments in force after damaging exercise are also influenced by a reduction in drive to the muscle (Morton et al., 2005).

Using the reliable (CV = 3.4%) twitch interpolation technique can provide an objective assessment of central drive by measuring VA (i.e. (1-[interpolated twitch force/resting twitch force]) x 100; Morton et al., 2005). For example, Prasartwuth et al. (2005) observed no change in in VA after eccentric exercise, whilst some have noted a reduced VA in the days after damaging exercise (Prasartwuth et al., 2006; Racinas et al., 2008; Behrens et al., 2012). Interestingly, Prasartwuth et al. (2006) noted that resting twitch force, MVC and VA were decreased up to eight days after eccentric exercise, but that the magnitude of the decrements was greater at shorter muscle lengths. Accordingly, they suggested that an increased central drive is required to activate a muscle at short length. Moreover, Sayers and colleagues (2003) noted that resting twitch force was impaired immediately and for up to nine days after eccentric exercise, suggesting that force decrements are due to peripheral factors such as damage to the contractile machinery (i.e. disruption of sarcomeres and impaired E-C coupling).

*2.7.2 Dynamic muscle function*

During sports movements, athletes will rarely ever perform isometric or isokinetic movements. Instead, muscle actions consist of a passive pre-stretch (eccentric phase) and an active shortening (concentric phase; Byrne et al., 2004; Komi, 2000; Ingen-Schenau et al., 1997). This is also known as the stretch-shortening cycle. This mechanism ultimately results in improved power output in the concentric phase (Ingen-Schenau et al., 1997). Vertical jump performance (drop jump, countermovement jump and squat jump) has been used extensively to assess the effect of EIMD on the stretch-shortening cycle.

Generally, studies have demonstrated reduced stretch-reflex sensitivity and muscle stiffness, resulting in diminished force producing capabilities (Byrne & Eston, 2001; Komi, 2000). The time course to recovery often follows a bimodal pattern (Byrne & Eston, 2002a; Byrne et al., 2004; Dousett et al., 2007; Eston et al., 2003). That is, an initial decline in jump performance followed by an early recovery and a secondary decline, which is indicative of the inflammatory response (Byrne et al., 2004; Dousett et al., 2007). Importantly, the time-course of jump performance is dependent on the use of stretch-shortening cycle. For example, Byrne and Eston (2001) reported greater decrements in squat jump (no stretch-shortening cycle) performance than countermovement jump and drop jump after muscle-damaging exercise. These findings suggest that the stretch-shortening cycle may attenuate performance decrements which follow muscle damaging exercise (Byrne & Eston, 2001; Byrne et al., 2004). Interestingly, these jump measures are used as a more ecologically valid marker of sporting performance after muscle-damaging exercise than single-jointed dynamometry. However, the dynamic nature of sporting performance means that athletes will undergo both high velocity-low force (i.e. sprints) and low-velocity-high-

force (i.e. scrummaging) conditions (Haff & Nimphius, 2012). To the author's knowledge, no data exists regarding the recovery of such movements after muscle-damaging exercise.

### *2.7.3 Perceived muscle soreness*

Delayed onset of muscle soreness is the most commonly assessed marker (Warren et al., 1999), yet the mechanism for its appearance remains unclear. Sensations of muscle soreness could result from a complex interaction of damage to the muscle structure, disrupted calcium homeostasis, and sensitisation of nocioceptors from inflammatory cell infiltrates (Hyldahl & Hubal, 2014). However, studies reporting increased muscle soreness after eccentric exercise in the presence of limited inflammation in both animal (Hayashi et al., 2017) and human models (Yu, Liu, Carlsson, Thornell & Stal, 2013) challenge the origins of delayed onset muscle soreness. Delayed onset muscle soreness typically appears between 8 - 24 h after muscle-damaging exercise, peaks between 24 - 48 h and usually subsides within 96 h (Damas et al., 2016; Jones, Newham & Clarkson, 1987).

### *2.7.4 Creatine kinase (CK) activity*

The appearances of muscle-specific proteins such as plasma CK and serum myoglobin, that peak 2 - 6 days after the initial insult, are typically reported (Byrne et al., 2004; Hyldahl et al., 2014; Warren et al., 1999). Membrane damage caused by eccentric lengthening leads to increased membrane permeability and leaking of muscle proteins into circulation (Sorichter, Puschendorf & Mair, 1999). Circulating muscle-specific proteins do, however, show a poor temporal relationship with muscle function (Friden & Lieber, 2001) and most likely indicate that tissue damage has

occurred rather than an indication of its magnitude.

## 2.8 Effects of ageing on responses to muscle-damaging exercise

### *2.8.1 Muscular strength*

Despite the importance of measuring muscular function in assessing the magnitude of EIMD (Damas, et al., 2016; Paulsen et al., 2012; Warren et al., 1999), its use in the EIMD and ageing literature is both sparse and unclear. For example, some studies have noted greater losses in force in older compared to young populations (Chapman et al., 2008; Ploutz-Synder et al., 2001), others greater decrements in young compared to old (Lavender & Nosaka, 2006; Lavender & Nosaka, 2007), and even no differences between age groups (Buford et al., 2014; Fell et al., 2006; Gordon et al., 2017; Lavender & Nosaka, 2008). The reasons for such discrepancy between these studies are unclear, but might be explained by the physical activity/resistance training status of the participants. For example, Burford and colleagues (2014) observed a similar recovery of isometric plantar flexion strength in young (~23 years) and older (~76 years) adults after eccentric unilateral plantar flexion exercise when controlling for physical activity levels. Recent literature has similarly reported no differences in the recovery of isometric and isokinetic forces from knee extensor exercise in young (~22 years) and middle-aged (~47 years) recreationally resistance trained males (Gordon et al., 2017). Gordon et al. (2017) suggested that for those who resistance-train, the recovery of muscular strength is similar regardless of age. Observations of comparable satellite cell responsiveness after eccentric plantar flexor exercise in physically active young and middle-aged males (Burford et al., 2014) support this notion. Holistically, these data might indicate that force loss after muscle-damaging

exercise is only different between age groups when physically activity level is not accounted for.

*2.8.2 Central versus peripheral fatigue*

Losses in muscular strength/force after exercise might be the result of central (i.e. neural impairments and a reduction in excitability to the alpha motor-neuron; Avela et al., 1999; Horita et al., 1999; Morton et al., 2005) and/or peripheral fatigue and/or damage (i.e. disruption of sarcomeres and impaired E-C coupling (Allen et al., 2008; Doguet et al., 2016; Hubal et al., 2007). For example, Macdonald, Button, Drinkwater and Behm (2014) observed decrements in MVC after muscle-damaging squatting exercise that were accompainied by impairments in VA and rest twitch torque (i.e. peripheral alterations). Although older populations are known to have lower baseline VA values (Klass, Baudry & Duchateau 2007), no study has determined the time course of VA or resting twitch scores after muscle-damaging exercise.

The avaliable data on resistance exercise-induced central and peripheral fatigue between age groups is limited to two studies investigating the immediate post-exercise alterations in young (~25 years) and old (~75 years) males (Dalton et al., 2012; Dalton et al., 2015). Dalton et al. (2012) observed no differences in VA or resting twitch torque between these groups after slow ($60°\cdot s^{-1}$), moderate ($180°\cdot s^{-1}$) or unconstrained velocity knee extension exercise. Similarly, Dalton et al. (2015) noted a comparable reduction in VA after isotonic and isokintic knees extension in young and old males. However, resting twitch torque was subject to decrements in the young group but not in their older counterparts (Dalton et al., 2015). Though Dalton et al. (2015) did not explain this, the reductions in resting twitch torque would indicate disruption of sarcomeres and impaired E-C coupling (Allen et al., 2008; Doguet et al.,

2016; Hubal et al., 2007) in the young group that were not present in the older group. To the author's knowledge, there in no data comparing the recovery of VA or resting twitch torque after muscle-damage exercise in young and middle-aged males.

*2.8.3 Perceived muscle soreness*

Several studies have presented equivocal findings on age-related differences in muscle soreness after muscle damaging exercise (Clarkson & Dendrick, 1988; Chapman et al., 2008; Fell et al., 2006; Lavender & Nosaka, 2006; Lavender & Nosaka, 2008). For example, older adults (~64 to 70 years) have reported lower soreness than young (~25 years; Chapman et al., 2008) and middle-aged adults (~48 years; Lavender & Nosaka, 2008), despite having greater decrements in MVC after muscle-damaging exercise (Chapman et al., 2008). These data are in contrast to studies reporting no differences in reported pain in young (~22 years) versus middle-aged (~47 years; Gordon et al., 2017) and young (~23 years) versus older (~67 years) adults after eccentric exercise (Clarkson & Dendrick, 1988) and those examining veteran cyclists (~45 years) who reported greater soreness than young (~24 years) cyclists over three consecutive days of time-trial cycling, in the presence of EIMD, despite no differences in performance (Fell, Reaburn & Harrison, 2008).

*2.8.4 Creatine kinase (CK) activity*

Several studies have examined the CK response to muscle-damaging exercise in different age groups (Chapman et al., 2008; Clarkson & Dendrick, 1988; Fell et al., 2006; Lavender & Nosaka, 2006; Lavender & Nosaka, 2008; Manfredi et al., 1991; Toft et al., 2002). In all age groups CK concentrations peaked at 4 to 5 days after the initial exercise, with most studies finding no difference between groups (Chapman et

al., 2008; Clarkson & Dendrick, 1988; Fell et al., 2006; Lavender & Nosaka, 2008; Manfredi et al., 1991). Two studies have noted higher CK activity in young compared to older males (Lavender & Nosaka, 2006; Toft et al., 2002), though given the commentary above it is only important to note that these CK concentrations were significantly increased from baseline in both groups.

## 2.9 Conclusion

The world population is ageing and alongside this is a growing number of middle-aged (35 to 55 years) males who regularly take part in sport. However, the ageing process is accompanied by hormonal and neuromuscular changes which result in losses of muscle mass, strength and power and subsequently, an impaired sporting performance. One consistent finding within the scientific literature is that the lower-body appears to be more suscepitble to losses of muscle mass, strength and power, but the reasons for this are unclear. Typically, these characteristics between age groups are measured using isometric or isokinetic dynamometry or physical functioning tests (i.e. stair climbing, chair rising), none of which provide useful information to the middle-aged males that play sport or engage in resistance training. The advent of rotary encoders means that muscle function can be assessed in a more ecologically valid way, though no study has sought to determine the functional characteristics of middle-aged resistance trained males using this technology.

In addition, resistance training provides an effective method of offsetting the loss of muscle mass, strength and power with improvements in strength and power ranging from 25 to 187% and 4 to 36%, respectively. These improvements vary depending on the weekly frequency, duration and intensity of the resistance training programme. Consequently, it is important for applied practitioners to monitor the

internal and external load imposed on the ageing male. Though methods such as repetition velocity and sRPE appear to be particularly useful, there is no ‘gold standard’ approach to monitoring internal and external load. Importantly, the author is unaware of any research that has sought to compare the internal and external load between young and middle-aged males, despite its importance.

The acute effects of resistance training are manifested in symptoms of EIMD, which can impair performance. The notion that ageing is associated with a greater magnitude of damage and an impaired recovery time has not been reported consistently in the literature. In fact, some studies have noted that young males experience greater muscle-damage than their older counterparts. Typically, the body of literature in this area has only focused those older than 60 years. Thus, the magnitude of EIMD and time-to-recovery in those aged 35 to 55 is unknown. The findings from research of this kind would be highly applicable to athletes looking to prolong their careers and optimise their training.

# 3. The intra- and inter-day reliability of the FitroDyne as a measure of multi-jointed muscle function

## 3.1 Introduction

Resistance training is widely used by strength and conditioning practitioners to advance athletic performance. Moreover, acute responses to resistance exercise result in impaired muscle function as a consequence of EIMD (Burt et al., 2014; Burt & Twist, 2011; Byrne et al., 2001; Davies et al., 2008; Davies et al., 2011; Doncaster & Twist, 2012). Losses in muscle function for up to a week after resistance exercise of ~21% (~60 $N \cdot m^{-1}$) and ~28% (~140 $N \cdot m^{-1}$) have been reported in physically active males for upper (Davies et al., 2008) and lower-body (Doncaster & Twist, 2012), respectively. However, used over a more extended period of time, resistance training programmes provide performance-related benefits, including increased power production (Aagaard et al., 2002), muscle strength (Mazzett et al., 2000), improved body composition (Mazzetti et al., 2000), vertical jump height (Christou et al., 2006) and sprint speed (Christou et al., 2006). Resistance training is typically periodised over a period of time by manipulating training volume (repetitions x sets x load) to enhance physiological adaptation (Baker et al., 1994). Over 6-12 weeks, such programmes demonstrate improvements of ~11-25% in maximal strength (Baker et al., 1994) and ~4-29% in muscle power (Naclerio et al., 2013; Sayer & Gibson, 2010; Turbanski & Schmidtbleicher, 2010). When assessing resistance training adaptations, applied practitioners should be cognisant that strength (~7.7% or 14 $N \cdot m^{-1}$; Nicolas, Gauthier, Bessot, Moussay & Davenne, 2005) and power (~3.1% or 165 W; West, Cook, Beaven & Kilduff, 2014) are greater in the evening than morning due to diurnal fluctuations.

These changes in muscle function are routinely assessed by having the participant perform a MVC, against a fixed arm (isometric) or an arm moving at a constant angular velocity (isokinetic). Both approaches have been used successfully to quantify the extent of muscle damage and the time course to recovery (Burt et al., 2014; Burt & Twist, 2011; Byrne et al., 2001; Davies et al., 2008; Davies et al., 2011; Doncaster & Twist, 2012; Hirose et al., 2004) and adaptions to training (Young & Bilby, 1993). However, their use often limits the real-world application of such findings as they fail to replicate the multi-jointed and dynamic nature of sporting movements and resistance training (Falvo & Bloomer, 2006). Moreover, the non-involvement of the stretch-shortening cycle (Marcora & Miller, 2000) and poor to moderate relationship with measures of athletic performance (Marocra & Miller, 2000; Young & Bilby, 1993) further impacts on the 'real world' validity of isokinetic and isometric muscle tests.

While the use of single-jointed dynamometry might be owing to the absence of more sophisticated measurement tools, the emergence a decade ago of rotary encoders (e.g. FitroDyne) has enhanced the possibilities for assessing muscle function during multi-jointed movements. Rotary encoders are devices that convert the motion into an analogue reading (e.g. power or velocity) via a rotating wheel tether and have been used to assess muscle function (Fry et al., 2006). Despite a high level of reliability being reported for this device in quantifying muscle power during single-jointed (bicep curl) or ballistic (squat jump) exercise (Jennings et al., 2005) and bar velocity during bench press (Stock et al., 2011), no study has determined its reliability during more traditional, multi-jointed resistance exercises that are habitually used in strength and conditioning training. Furthermore, given the need to assess acute alterations in muscle function pre- and immediately post-intervention (i.e. intra-day; Byrne et al., 2001), the authors are unaware of any such study that has established

the intra-day reliability of upper-body pushing and pulling movements. Whether for athlete support or inclusion in research, the importance of certifying that a measurement tool is reliable is acknowledged (Atkinson & Nevil, 1998). By determining both the intra- and inter-day reliability it can be accepted that an instrument is capable of detecting changes through interventions and not technical error or biological variation. As such, the purpose of this study is to assess the intra- and inter-day reliability of the FitroDyne during bench press, squat and bent-over-row movements as measures of multi-jointed muscle function.

## 3.2 Methods

### *3.2.1 Participants*

Fourteen healthy males (see Table 1) were recruited to the study via convenience sampling. All were asymptomatic of illness and injuries, had resistance trained for at least two years and were accustomed to the exercises used. The participants provided written informed consent and the study was granted approval from the Research Ethics Committee of the Faculty of Life Sciences.

**Table 3.1** Participant characteristics.

| Characteristic | Mean ± SD |
|---|---|
| **Age (y)** | 22.6 ± 4.9 |
| **Mass (kg)** | 83.2 ± 8.1 |
| **Stature (m)** | 1.8 ± 0.1 |
| **Fat mass (kg)** | 10.0 ± 4.3 |
| **Fat free mass (kg)** | 73.2 ± 7.4 |
| **Bench press 1RM (kg)** | 102.5 ± 19.0 |
| **Squat 1RM (kg)** | 132.2 ± 26.2 |
| **Bent-over-row 1RM (kg)** | 94.8 ± 14.5 |

### *3.2.2 Study design*

The study incorporated a repeated measures design and was a conceptual replication of Jennings et al. (2005). Participants first attended the laboratory for a familiarisation trial, during which anthropometric measurements (stature, body mass and body composition prediction) were followed by multiple resistance trials at various loads of the selected exercise performed until their power values plateaued (Batterham & George, 2003). That is, they were considered to be 'familiarised' when they could complete three successive repetitions that yielded power values within 10% of each other. The participants returned to the laboratory 48 hours later for trial 1 in which they completed three repetitions of bench press, squat and bent-over-row at various loads. Replica trials - 2 and 3 - were conducted two and 48 hours later, respectively.

### *3.2.3 Procedures*

#### *3.2.3.1 Strength testing*

Participants' maximum strength on the bench press and bent-over-row exercises were assessed by a standardised direct 1RM protocol, as in Stock et al. (2011). One repetition maximum for squat exercise was predicted via a 5RM protocol, for safety reasons, in the manner outlined by Reynolds, Gordon and Robergs (2006), and from the equation:

$$1RM\ (kg) = 1.0970 \times (5RM\ weight\ [kg]) + 14.2546$$

The above equation was reported to yield accurate 1RM predictions ($R^2$ = 0.974, SEE = 13.51 kg) by Reynolds et al. (2006).

*3.2.3.2 Assessment of muscle function*

During the three repeated trials, peak and mean power and velocity were assessed on the three exercises at loads corresponding to 20, 30, 40, 50, 60, 70 and 80% of the 1RM values in a randomised order via the FitroDyne apparatus (Fitronic, Bratislava, Slovakia) attached directly under a bearing-supported linear raise Smith machine bar (Smith Machine standard, Perform Better, Leicester, UK) by its nylon cable (< 2 N resistance). The FitroDyne measures rate of displacement (at 100 Hz) and thus assumes that the nylon cord is moving in a vertical plane. Any deviation from this plane could increase measurement error. As such the Smith machine, with a 20 kg barbell, was employed as it restricts the movement of the nylon cord to the vertical plane only.

For the bench press exercise, the participant held the bar with a prone grip and lowered it to his chest in a controlled manner, before maximally pushing until full elbow extension. For the squat exercise, the bar was positioned across the shoulders. Participants descended until their hips were below the knee joint and ascended as rapidly as possible until their knees were at full extension. A bench was employed to ensure that they attained the same depth and range of motion on each repetition. Muscle function for the bent-over-row exercise was determined with the participant commencing in a bent-over position, before pulling the bar maximally until the elbows reached full flexion. Three repetitions of each exercise were performed at each load with self-selected rest intervals that were capped at 90 s, but ranged from 30-90 s (Jennings et al., 2005). Rest times were self-selected, as lighter loads did not require the same recovery time. Peak and mean velocity values recorded from these trials were used in the data analysis. The exercise and load sequence was randomised for each participant to negate possible order effects.

*3.2.4 Statistical analysis*

All data collected were analysed using SPSS (version 21, IBM SPSS Inc, Chicago, IL). Peak and mean values for power and velocity for the three repetitions at each load were used in the assessment of the intra- (trial 1 versus trial 2) and inter-day (trial 1 versus trial 3) reliability of the FitroDyne. The assumption of normality of the distributions of the dependent variables was checked via the Shapiro-Wilk statistic and found to be satisfied ($P > 0.05$). Accordingly, a one-way repeated measures analysis of variance (ANOVA) was conducted to test for systematic error between the trials. Alpha was set at 0.05.

Having established that the differences (errors) were found to be homoscedastic, the trial-to-trial reliability of the FitroDyne data was quantified via the typical error (TE; standard deviation of the differences divided by $\sqrt{2}$), CV (TE divided by the grand mean test-retest score, multiplied by 100) statistics, as described by Hopkins (2000). It has been argued that TE is preferable to 95% limits of agreement (95% LoA) technique as the latter is too stringent to detect meaningful changes in sports/exercise performance (Hopkins, 2000). Moreover, the smallest worthwhile change (SWC; 0.2 multiplied by the shared standard deviation) and moderate change (MC; SWC multiplied by 3) were calculated to provide a ‘real world’ application of the findings. To detect genuine training-related reductions in muscle function via multi-jointed measures, the dependent variables were considered capable of detecting small or moderate changes if the TE was smaller than the SWC or MC, respectively (Pyne, 2003).

### 3.3 Results

The descriptive statistics for the muscle function variables of each exercise across the three trials (at each load) are presented in Figures 3.1 to 3.3. ANOVA revealed no significant ($P > 0.05$) bias in each variable between trials 1 and 2 at any load. For all the dependent variables and exercises the TE was greater than the SWC, but smaller than the MC intra-day across all loads (Tables 3.2 to 3.4). The best intra-day reliability for bench press was noted for peak power at 40% 1RM (TE and CV = 10.1 W and 1.6 %, respectively). The lowest levels of reliability were displayed for the intra-day bench press at the 80% intensity for peak and mean power and mean velocity, with TEs of 53 W (CV = 12.2%) and 44.2 W (CV = 17.1%) and 4.3 (CV = 13.4%) $cm \cdot s^{-1}$, respectively. Squat across all loads for peak power, mean power, peak velocity and mean velocity displayed TEs of 21.8-73.3 W (CV = 2.4-6.1%), 8.7-29.6 W (CV = 2.4-6.4 %), 3.9-8.1 $cm \cdot s^{-1}$ (CV = 2.7-5.8%) and 2-4.2 $cm \cdot s^{-1}$ (CV = 2.2-6.4%), respectively. For bent-over-row, the intra-day reliability was generally better, with the poorest levels of agreement observed for mean velocity at 70%1RM (TE and CV = 6.7 $cm \cdot s^{-1}$ and 9%, respectively).

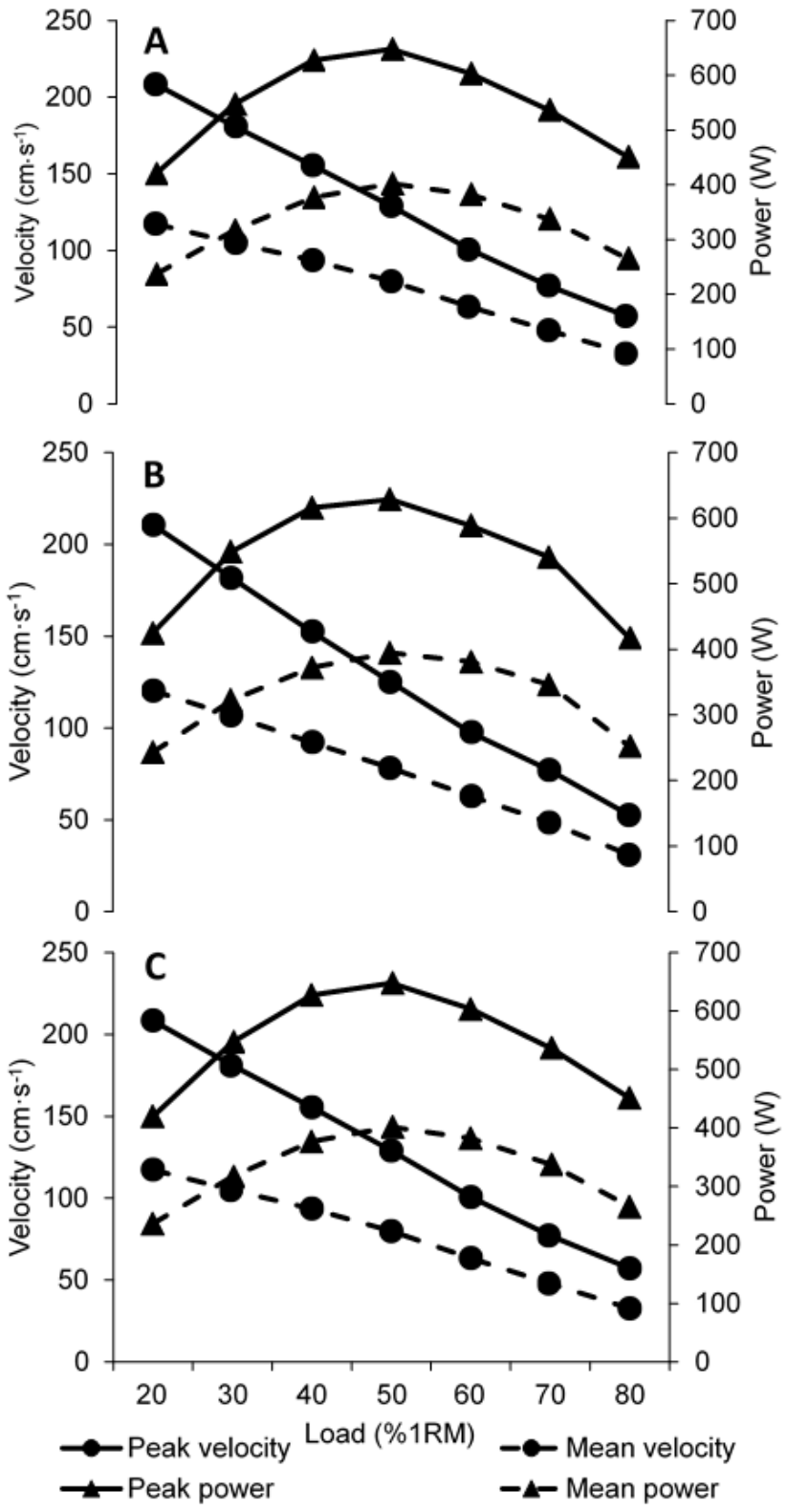


**Figure 3.1** Sample mean values for peak power, mean power, peak velocity and mean velocity for bench press during trials 1 (A), 2 (B) and 3 (C).

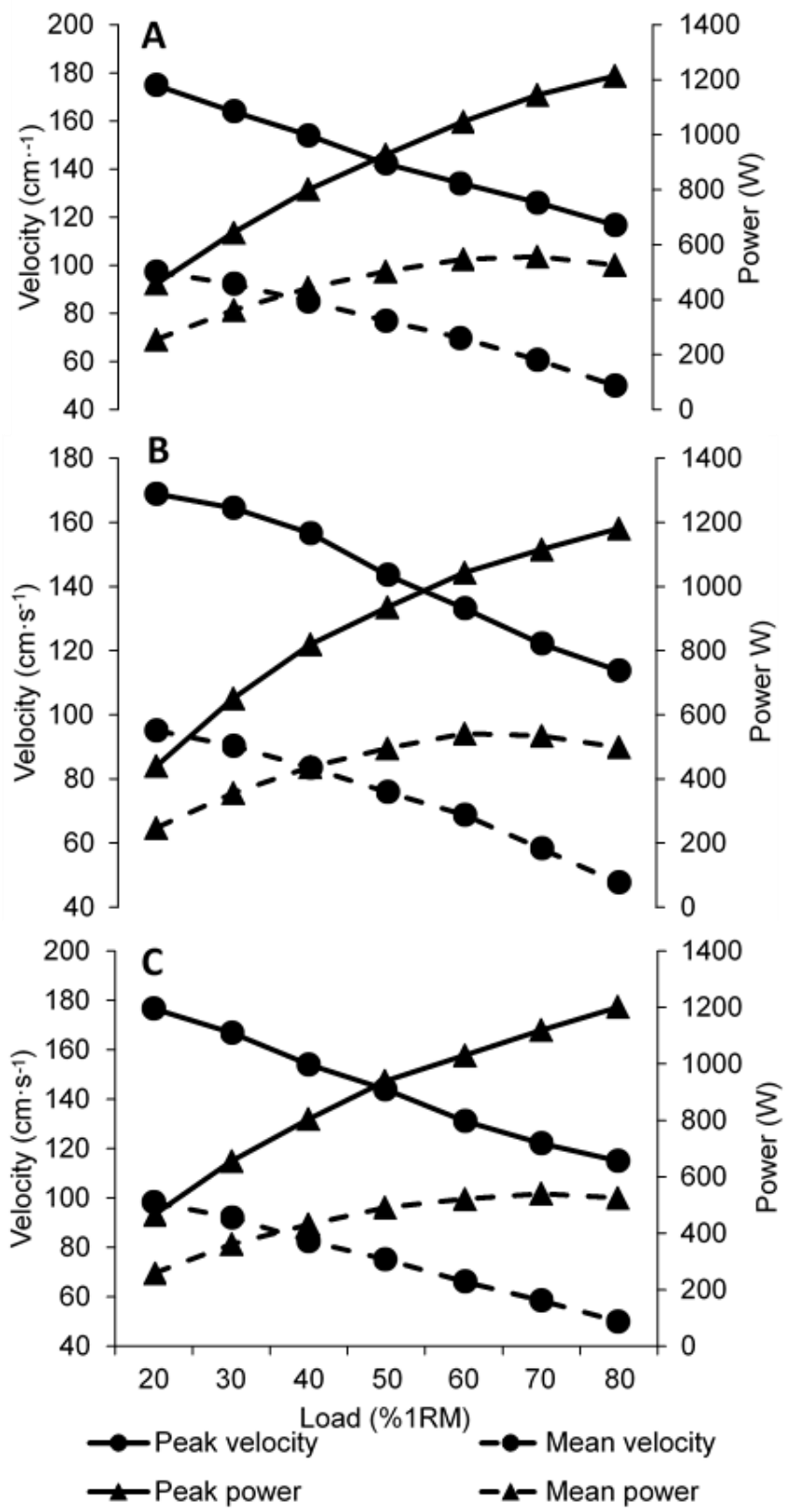


**Figure 3.2** Sample mean values for peak power, mean power, peak velocity and mean velocity for squat during trials 1 (A), 2 (B) and 3 (C).

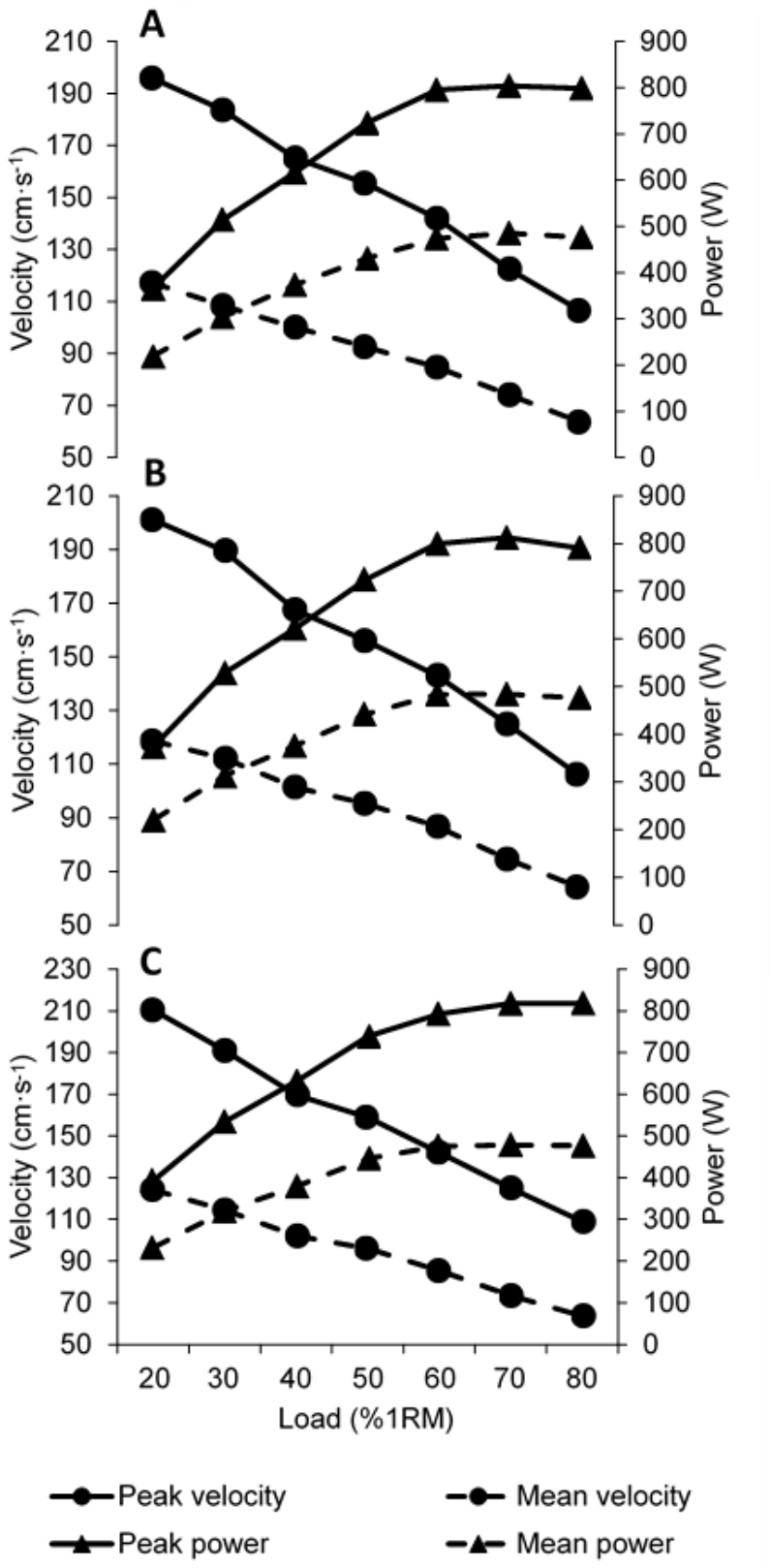


**Figure 3.3** Sample mean values for peak power, mean power, peak velocity and mean velocity for bent-over-row during trials 1 (A), 2 (B) and 3 (C).

Similarly, ANOVA revealed no significant bias ($P > 0.05$) for any exercise, load or variable between trials 1 and 3. For the inter-day reliability, the TE for peak power, mean power, peak velocity or mean velocity, was unable to detect the SWC across any exercise or load, but was able to identify the MC (Tables 3.2 to 3.4). Bench press demonstrated similar inter-day reliability to intra-day reliability at loads of 20-70%1RM. Interestingly, inter-day reliability for peak and mean power and mean velocity at 80% was better, with TEs of 32.1 (CV = 7.1%) and 22.5 W (CV = 8.6%) and 2.6 (CV = 8.2%) $cm \cdot s^{-1}$, respectively. Inter-day reliability for squat was similar to the intra-day results for all dependent variables across all loads, while for bent-over-row it was comparable to the intra-day reliability across all loads for peak power, mean power, peak velocity and mean velocity.

**Table 3.2** Reliability statistics for peak and mean power and velocity during bench press exercise.

| | | Peak Power | | | | Mean Power | | | | Peak Velocity | | | | Mean Velocity | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Load (%1RM)** | **Trial** | **TE (W)** | **SWC (W)** | **MC (W)** | **CV (%)** | **TE (W)** | **SWC (W)** | **MC (W)** | **CV (%)** | **TE ($cm \cdot s^{-1}$)** | **SWC ($cm \cdot s^{-1}$)** | **MC ($cm \cdot s^{-1}$)** | **CV (%)** | **TE ($cm \cdot s^{-1}$)** | **SWC ($cm \cdot s^{-1}$)** | **MC ($cm \cdot s^{-1}$)** | **CV (%)** |
| **20** | *1 v 2* | 18.3 | 7.7 | 23.2 | 4.3 | 8.2 | 3.5 | 10.5 | 3.4 | 6.9 | 2.9 | 8.8 | 3.3 | 3.4 | 1.5 | 4.4 | 2.9 |
| | *1 v 3* | 17.0 | 7.2 | 21.7 | 4.0 | 13.8 | 5.9 | 17.6 | 5.7 | 6.7 | 2.8 | 8.5 | 3.2 | 5.1 | 2.2 | 6.5 | 4.3 |
| **30** | *1 v 2* | 11.3 | 4.8 | 14.3 | 2.1 | 8.4 | 3.6 | 10.7 | 2.6 | 3.1 | 1.3 | 3.9 | 1.7 | 2.9 | 1.2 | 3.7 | 2.7 |
| | *1 v 3* | 14.1 | 6.0 | 17.9 | 2.6 | 13.1 | 5.6 | 16.7 | 4.1 | 4.1 | 1.7 | 5.2 | 2.2 | 3.7 | 1.6 | 4.7 | 3.5 |
| **40** | *1 v 2* | 10.1 | 4.3 | 12.8 | 1.6 | 11.0 | 4.7 | 14.0 | 2.9 | 2.8 | 1.2 | 3.6 | 1.8 | 2.9 | 1.2 | 3.7 | 3.1 |
| | *1 v 3* | 13.4 | 5.7 | 17.1 | 2.2 | 11.5 | 4.9 | 14.6 | 3.1 | 3.3 | 1.4 | 4.1 | 2.1 | 2.8 | 1.2 | 3.6 | 3.0 |
| **50** | *1 v 2* | 16.9 | 7.2 | 21.5 | 2.6 | 10.7 | 4.5 | 13.6 | 2.7 | 3.6 | 1.5 | 4.5 | 2.8 | 2.2 | 1.0 | 2.9 | 2.8 |
| | *1 v 3* | 28.0 | 11.9 | 35.7 | 4.4 | 18.6 | 7.9 | 23.6 | 4.7 | 5.8 | 2.5 | 7.4 | 4.6 | 4.0 | 1.7 | 5.1 | 5.1 |
| **60** | *1 v 2* | 23.8 | 10.1 | 30.3 | 4.0 | 17.7 | 7.5 | 22.5 | 4.6 | 3.8 | 1.6 | 4.9 | 3.9 | 2.8 | 1.2 | 3.5 | 4.4 |
| | *1 v 3* | 27.8 | 11.8 | 35.4 | 4.6 | 16.2 | 6.9 | 20.7 | 4.2 | 4.3 | 1.8 | 5.5 | 4.3 | 2.4 | 1.0 | 3.1 | 3.8 |
| **70** | *1 v 2* | 18.6 | 7.9 | 23.6 | 3.4 | 16.4 | 7.0 | 20.9 | 4.8 | 2.6 | 1.1 | 3.2 | 3.3 | 2.2 | 0.9 | 2.8 | 4.5 |
| | *1 v 3* | 34.2 | 14.5 | 43.5 | 6.4 | 21.6 | 9.2 | 27.5 | 6.4 | 4.5 | 1.9 | 5.7 | 5.9 | 3.0 | 1.3 | 3.8 | 6.2 |
| **80** | *1 v 2* | 53.0 | 22.5 | 67.5 | 12.2 | 44.2 | 18.7 | 56.2 | 17.1 | 5.3 | 2.3 | 6.8 | 9.7 | 4.3 | 1.8 | 5.4 | 13.4 |
| | *1 v 3* | 32.1 | 13.6 | 40.8 | 7.1 | 22.5 | 9.6 | 28.7 | 8.6 | 3.4 | 1.5 | 4.4 | 6.1 | 2.6 | 1.1 | 3.3 | 8.2 |

**Table 3.3** Reliability statistics for peak and mean power and velocity during squat exercise.

| | | Peak Power | | | | Mean Power | | | | Peak Velocity | | | | Mean Velocity | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Load (%1RM)** | **Trial** | **TE (W)** | **SWC (W)** | **MC (W)** | **CV (%)** | **TE (W)** | **SWC (W)** | **MC (W)** | **CV (%)** | **TE ($cm \cdot s^{-1}$)** | **SWC ($cm \cdot s^{-1}$)** | **MC ($cm \cdot s^{-1}$)** | **CV (%)** | **TE ($cm \cdot s^{-1}$)** | **SWC ($cm \cdot s^{-1}$)** | **MC ($cm \cdot s^{-1}$)** | **CV (%)** |
| **20** | *1 v 2* | 21.8 | 9.2 | 27.7 | 4.9 | 13.3 | 5.6 | 16.9 | 5.3 | 7.0 | 3.0 | 9.0 | 4.1 | 3.9 | 1.6 | 4.9 | 4.0 |
| | *1 v 3* | 22.5 | 9.6 | 28.7 | 4.9 | 11.5 | 4.9 | 14.7 | 4.5 | 6.5 | 2.8 | 8.3 | 3.7 | 3.9 | 1.7 | 5.0 | 4.0 |
| **30** | *1 v 2* | 34.6 | 14.7 | 44.1 | 5.4 | 19.9 | 8.4 | 25.3 | 5.6 | 7.4 | 3.1 | 9.4 | 4.5 | 4.2 | 1.8 | 5.4 | 4.6 |
| | *1 v 3* | 27.6 | 11.7 | 35.2 | 4.3 | 8.7 | 3.7 | 11.1 | 2.4 | 5.9 | 2.5 | 7.6 | 3.6 | 2.0 | 0.9 | 2.6 | 2.2 |
| **40** | *1 v 2* | 35.2 | 14.9 | 44.8 | 4.3 | 28.2 | 12.0 | 35.9 | 6.4 | 6.1 | 2.6 | 7.8 | 4.0 | 4.8 | 2.0 | 6.1 | 5.7 |
| | *1 v 3* | 35.5 | 15.0 | 45.1 | 4.4 | 16.5 | 7.0 | 21.0 | 3.8 | 6.2 | 2.6 | 7.9 | 4.0 | 3.3 | 1.4 | 4.3 | 4.0 |
| **50** | *1 v 2* | 22.8 | 9.7 | 29.0 | 2.4 | 14.2 | 6.0 | 18.1 | 2.9 | 3.9 | 1.7 | 5.0 | 2.7 | 2.4 | 1.0 | 3.1 | 3.2 |
| | *1 v 3* | 54.3 | 23.0 | 69.0 | 5.8 | 24.3 | 10.3 | 31.0 | 4.9 | 8.1 | 3.4 | 10.3 | 5.7 | 4.2 | 1.8 | 5.3 | 5.5 |
| **60** | *1 v 2* | 36.7 | 15.6 | 46.7 | 3.5 | 22.3 | 9.4 | 28.3 | 4.1 | 4.5 | 1.9 | 5.7 | 3.3 | 3.0 | 1.3 | 3.8 | 4.3 |
| | *1 v 3* | 54.9 | 23.3 | 69.9 | 5.3 | 22.4 | 9.5 | 28.6 | 4.2 | 7.0 | 3.0 | 8.9 | 5.3 | 3.1 | 1.3 | 3.9 | 4.5 |
| **70** | *1 v 2* | 55.6 | 23.6 | 70.8 | 4.9 | 29.6 | 12.6 | 37.7 | 5.4 | 6.4 | 2.7 | 8.2 | 5.2 | 3.8 | 1.6 | 4.8 | 6.3 |
| | *1 v 3* | 64.6 | 27.4 | 82.2 | 5.7 | 21.9 | 9.3 | 27.9 | 4.0 | 6.9 | 2.9 | 8.8 | 5.6 | 2.5 | 1.1 | 3.2 | 4.2 |
| **80** | *1 v 2* | 46.7 | 19.8 | 59.5 | 3.9 | 28.4 | 12.1 | 36.2 | 5.5 | 5.1 | 2.2 | 6.5 | 4.4 | 3.1 | 1.3 | 4.0 | 6.4 |
| | *1 v 3* | 73.3 | 31.1 | 93.3 | 6.1 | 23.4 | 9.9 | 29.7 | 4.4 | 6.8 | 2.9 | 8.6 | 5.8 | 2.6 | 1.1 | 3.3 | 5.2 |

**Table 4.** Reliability statistics for peak and mean power and velocity during bent-over-row exercise.

| | | Peak Power | | | | Mean Power | | | | Peak Velocity | | | | Mean Velocity | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Load (%1RM)** | **Trial** | **TE (W)** | **SWC (W)** | **MC (W)** | **CV (%)** | **TE (W)** | **SWC (W)** | **MC (W)** | **CV (%)** | **TE ($cm \cdot s^{-1}$)** | **SWC ($cm \cdot s^{-1}$)** | **MC ($cm \cdot s^{-1}$)** | **CV (%)** | **TE ($cm \cdot s^{-1}$)** | **SWC ($cm \cdot s^{-1}$)** | **MC ($cm \cdot s^{-1}$)** | **CV (%)** |
| **20** | *1 v 2* | 18.9 | 8.0 | 24.0 | 5.1 | 14.5 | 6.2 | 18.5 | 6.6 | 8.8 | 3.7 | 11.2 | 4.4 | 6.6 | 2.8 | 8.5 | 5.6 |
| | *1 v 3* | 15.2 | 6.4 | 19.3 | 4.0 | 10.3 | 4.4 | 13.1 | 4.6 | 7.6 | 3.2 | 9.6 | 3.7 | 5.4 | 2.3 | 6.8 | 4.5 |
| **30** | *1 v 2* | 20.0 | 8.5 | 25.5 | 3.8 | 23.0 | 9.7 | 29.2 | 7.4 | 7.1 | 3.0 | 9.0 | 3.8 | 8.7 | 3.7 | 11.1 | 7.9 |
| | *1 v 3* | 15.1 | 6.4 | 19.2 | 2.9 | 17.2 | 7.3 | 21.9 | 5.5 | 5.9 | 2.5 | 7.5 | 3.2 | 7.0 | 3.0 | 8.9 | 6.3 |
| **40** | *1 v 2* | 21.6 | 9.2 | 27.5 | 3.5 | 16.3 | 6.9 | 20.7 | 4.3 | 6.1 | 2.6 | 7.7 | 3.7 | 4.8 | 2.0 | 6.1 | 4.7 |
| | *1 v 3* | 24.1 | 10.2 | 30.7 | 3.9 | 20.4 | 8.6 | 25.9 | 5.4 | 4.9 | 2.1 | 6.3 | 2.9 | 4.7 | 2.0 | 6.0 | 4.7 |
| **50** | *1 v 2* | 20.8 | 8.8 | 26.5 | 2.9 | 26.7 | 11.3 | 33.9 | 6.1 | 4.0 | 1.7 | 5.1 | 2.6 | 6.0 | 2.6 | 7.7 | 6.4 |
| | *1 v 3* | 18.6 | 7.9 | 23.7 | 2.6 | 25.8 | 11.0 | 32.9 | 5.9 | 3.7 | 1.6 | 4.7 | 2.3 | 5.8 | 2.5 | 7.4 | 6.2 |
| **60** | *1 v 2* | 33.0 | 14.0 | 42.1 | 4.1 | 33.0 | 14.0 | 42.0 | 6.9 | 5.7 | 2.4 | 7.2 | 4.0 | 6.4 | 2.7 | 8.1 | 7.5 |
| | *1 v 3* | 41.0 | 17.4 | 52.2 | 5.2 | 34.9 | 14.8 | 44.4 | 7.3 | 5.9 | 2.5 | 7.5 | 4.1 | 6.3 | 2.7 | 8.0 | 7.4 |
| **70** | *1 v 2* | 62.8 | 26.7 | 80.0 | 7.8 | 40.0 | 17.0 | 50.9 | 8.2 | 10.5 | 4.4 | 13.3 | 8.5 | 6.7 | 2.8 | 8.5 | 9.0 |
| | *1 v 3* | 53.3 | 22.6 | 67.8 | 6.6 | 33.0 | 14.0 | 42.0 | 6.8 | 8.0 | 3.4 | 10.1 | 6.4 | 4.8 | 2.1 | 6.2 | 6.6 |
| **80** | *1 v 2* | 61.1 | 25.9 | 77.8 | 7.7 | 37.3 | 15.8 | 47.4 | 7.8 | 8.8 | 3.7 | 11.2 | 8.3 | 5.4 | 2.3 | 6.9 | 8.5 |
| | *1 v 3* | 68.8 | 29.2 | 87.5 | 8.5 | 28.8 | 12.2 | 36.6 | 6.0 | 9.5 | 4.0 | 12.0 | 8.8 | 3.7 | 1.6 | 4.7 | 5.9 |

### 3.4 Discussion

This study has observed that measures of muscle function assessed via the FitroDyne can be reproduced within acceptable limits both intra- and inter-day. Importantly, they suggest that the FitroDyne can be used with confidence to monitor moderate changes among athletes during multi-jointed exercise, either in detecting fatigue, muscle damage or as a result of training adaptation, independently of an athlete's power.

The threshold of reliability of a measurement tool is dependent on the setting it is applied in (Atkinson & Nevil, 1998). After muscle-damaging exercise acute decrements in muscle function ranging from 14.5-28.2% are typically observed (Burt et al., 2014; Burt & Twist, 2011; Davies et al., 2008; Davies et al., 2011), while increases in muscle strength and muscle power after resistance training are 11-25% and 4-29%, respectively (Baker, 1994; Naclerio et al., 2013; Sayers & Gibson, 2010; Turbanski & Schmidtbeicher, 2010). Accordingly, a variation of up to 10% would allow for suitable detection of changes in muscle function in these settings. Though, this emphasises the CV%, the TE, SWC and MC should be incorporated to support the interpretation of the reliability data. Few studies adopt 'analytical goals', but their consideration adds value to the analysis of the findings (Highton et al., 2012).

These data have demonstrated that intra-day the FitroDyne can detect moderate, but not small, changes in power and velocity for bench press. Comstock and colleagues (Comstock et al., 2011) noted an intra-class correlation coefficient of ≥ 0.96 for bench throw at 30% 1RM. For comparative purposes, the bench press intra-day intra-class correlation coefficient in this study was 0.98. Moreover, peak and mean power and velocity were deemed reliable at loads up to 70% 1RM. At 80% 1RM, however, peak and mean power and mean velocity displayed unacceptable intra-day reliability (TEs of 53 and 44.2 W and 4.3 $cm \cdot s^{-1}$, respectively) as all CV% were greater

than the analytical goal. Bench press peak velocity intra-day reliability was better, at 9.7 CV%, but close to the threshold.

Such low intra-day reliability for bench press at 80% 1RM might reflect the presence of fatigue from trial 1. That is to say, the recovery from trials 1 to 2 was insufficient to enable the restoration of muscle function, particularly for low velocity, high force movements. That this study observed larger, albeit not significant decrements in velocity and power at 80% 1RM from trials 1 to 2 compared to 1 to 3, supports this notion. Slower movement velocities (80 and 90% 1RM) have also been reported to possess the poorest reproducibility (Stock et al., 2011). While the reasons are not entirely clear, Stock et al. (2011) speculated that the FitroDyne device might not be able to detect small differences in slow movement velocities. The applied practitioner should be confident in identifying moderate changes in upper pushing power and velocity at 20-70% 1RM, but not at 80% 1RM.

Small intra-day TE and CV% for squat exercise velocity (2-8.1 $cm \cdot s^{-1}$ and 2.2-6.4%, respectively) and power (8.7-73.3 W and 2.4-6.4%, respectively) suggested that the movement was repeatable. These findings are consistent with Cormack et al. (2008) who, like this study, noted that intra-day TE was not able to detect the SWC and produced CVs of 3.5 and 6.9% for peak and mean power, respectively. During squat jump exercise at 30% 1RM Comstock et al. (2011) observed an intra-day intra-class correlation coefficient of ≥ 0.96, similar to the current study's intra-class correlation coefficient of 0.95 at 30% 1RM. Regarding the application of these findings, Byrne and colleagues (2001) quantified decrements of 23.2 and 29.7% in isokinetic torque at slow (0.52 $rad \cdot s^{-1}$) and fast (3.14 $rad \cdot s^{-1}$) angular velocities, respectively. At all exercise loads, the CV%s for power and velocity reflect that the FitroDyne would be able to detect these changes observed by Byrne et al. (2001).

Until now no study has assessed the intra-day reliability of upper-body pulling power or velocity. This study reports a high degree of reliability for the assessment of power and velocity during bent-over-row exercise across a range of loads. These findings suggest that bent-over-row can be used with confidence to assess moderate intra-day changes in an applied setting. Similar to bench press and squat, the FitroDyne can be used to quantify moderate changes in pulling exercise after muscle-damaging exercise. However, the paucity of research on pulling exercises means it is difficult to relate such reliability to known power or velocity changes after resistance training.

The *inter*-day reliability for bench press peak and mean power and velocity was generally similar to the intra-day reliability at loads of 20-70% 1RM, but superior at 80% 1RM (TE of 32.1 and 22.5 W and 2.6 $cm \cdot s^{-1}$ and CVs of 7.1, 8.6 and 8.2% for peak and mean power and mean velocity, respectively), though in all cases the TE was only able to detect the MC. This enhanced reliability might be due to sufficient time to off-set fatigue, illustrated in the restoration of velocity at this load. During bench press Stock et al. (2001) observed CVs of 3.1 and 12.6% for inter-day peak velocity at 10-90% 1RM. Drinkwater et al. (2005) found that a TE of 14 W, at 40 kg (no relative load noted), was not able to detect the SWC. After eight weeks of strength training Turbanski and Schmidtbleicher (2010) noted a 10 $cm \cdot s^{-1}$ (4.2%) improvement in maximum velocity during bench press exercise. This change might be detectable using the FitroDyne as the TE calculated in this study was 6.7 $cm \cdot s^{-1}$ (CV = 3.2%) at 20% 1RM, which produced the fastest velocity. Furthermore, a 37.3 W (15.5%) improvement in bench press power during a progressive resistance test has been noted following higher volume resistance training. While there are no direct load comparisons, the worst TE and CV (34.2 W and 7.1%) are low enough to detect the

aforementioned improvements. Additionally, acute decrements of 28.2 and 21.9% (140.4 and 108.9 $N \cdot m^{-1}$; Aagaard et al., 2002) in pushing force 24 and 48 hours post muscle-damaging exercise are within the threshold of the FitroDyne's measurement error.

Inter-day reliability for squat exercise was, as for bench press, similar to intra-day. The TEs for power and velocity of 8.7-73.3 W (CV = 2.4-6.1%) and 2.0-8.1 $cm \cdot s^{-1}$ (CV = 2.2-5.8%) respectively, reinforced the FitroDyne's ability to detect moderate changes. Such interpretations, of 'good reliability' agree with previous reports, albeit the statistics are not directly comparable (Jennings et al., 2005; Comstock et al., 2011; Cormack et al., 2008). For example, Jennings et al. (2005) reported an intra-class correlation coefficient of 0.97 and 95% LoA of -17 ± 96 W for squat jump peak power, whilst we noted an intra-class correlation coefficient of 0.91 and 95% LoA of 11.1 ± 271 W (not included in results) for peak power. As decrements in lower-limb muscle function are large (14.5-20.9%; Burt et al., 2014; Burt & Twist, 2011; Davies et al., 2011; Davies et al., 2008), the current findings indicate the FitroDyne is capable of detecting such changes across a range of exercise loads. However, the typically small (5.6%) improvement in muscle power during squat following six weeks resistance training (Naclerio et al., 2013) is lower than the TE and CV% and therefore challenges the FitroDyne's sensitivity to detect this.

With respect to bent-over-row exercise, our findings reflect levels of reliability – TE and CVs for power (10.3-68.8 W and 2.6-8.5%) and velocity (3.7-9.5 $cm \cdot s^{-1}$ and 2.3-8.8%) – that were mostly low and sufficient to detect moderate changes in muscle function. Though there are no reports of acute muscle functional changes for this exercise after resistance training, the study by Naclerio et al. (2013) involving six weeks of resistance training yielded a 37.3 W (13.5%) improvement in upright row

pulling power during a progressive resistance test. Despite mechanical differences of upright rows when compared to bent-over-row, the TEs in our study are too high to detect such adaptations. As with bench press and squat, it is recommended that the FitroDyne be used to assess muscle-damaging exercise provided the expected changes are not less than moderate.

Applied practitioners should be mindful that these findings were observed on a fixed vertical plane of motion during Smith machine exercise. The Smith machine was employed in order to avoid any deviation from a vertical plane as this could increase measurement error. Consequently, the reliability noted might not always be representative of free weight barbell movements. Future research might seek to determine if measurement error differs between resistance exercises performed on a Smith machine compared to free weight barbell apparatus.

### 3.5 Conclusion

This is the first study, to our knowledge, to provide a comprehensive appraisal of the reliability of muscle function measures during traditional multi-joint resistance exercises using a commercially available rotary encoder. The device's intra-day reliability indicated it could detect moderate, but not small, changes for all exercises and loads for peak and mean power and velocity. For bench press, intra-day reliability for loads up to 70% 1RM was good, but less so at 80% 1RM, possibly owing to the associated low velocity, or acute fatigue from the previous testing protocol. Squat and bent-over-row intra-day reliability was good throughout. These data support the use of the FitroDyne to quantify acute intra-day changes in muscle function. Inter-day reliability was similar to intra-day for all exercises, and better at the highest tested intensity (80% 1RM) for bench press. Overall, our findings suggest that the FitroDyne

can be used with confidence to assess muscle function during traditional resistance training exercise and to measure acute changes in muscle function across a range of submaximal loads. The applied practitioner should, however, be cautious when assessing muscle-function intra-day at 80% 1RM during bench press exercise. In addition, the FitroDyne provides an alternative to the use of single-jointed isometric and isokinetic dynamometry in the assessment of muscle function.

# 4. A comparison of load-velocity and load-power relationships between well-trained young and middle-aged males during three popular resistance exercises

## 4.1 Introduction

Current demographic trends indicate that the number of middle-aged people (30 to 59-year-olds) in the U.K. is increasing (Office for National Statistics, 2014), with the expectation that the number will rise from 25.7 million in 2014 to 26.3 million in 2020. Improvements in medical care and a greater appreciation for factors that enhance longevity are said to contribute to these demographic changes (Baker & Tang, 2010). Alongside this transformation is the growing number of middle-aged athletes (Lepers et al., 2013; Tanaka & Seals, 2008), who, despite the inevitable declines in athletic ability owing to the ageing process (Anton et al., 2004; Baker & Tang, 2010; Baker, Tang & Turner, 2003; Tanaka & Seals, 2008), strive to maintain or improve their athletic performance. Examples of such age-related declines have been documented by Pantoja, Villareal, Brisswalter, Peyre-Tartaruga and Morin (2016) who reported an ~ 1% decline per year between the ages of 39 and 96 years in maximal velocity and power outputs during a 30 m sprint, and by Baker and Tang (2010) who noted a 25% difference in weightlifting performances (World Records) of those in 30- and 60-year-old age categories.

Impairment in athletic performance as an athlete ages is largely due to the age-associated changes in muscle quality illustrated in muscle atrophy (i.e. sarcopenia; Narici, Reeves, Morse & Maganaris, 2004; Welle, 2002) and a loss of muscle strength and power (i.e. dynapenia; Candow & Chilibeck, 2007; Izqueirdo et al., 1999; Clark & Manini, 2012). Early work by Frontera and colleagues (1991) suggested that

sarcopenia was a major factor when explaining dynapenia. However, more recent longitudinal and cross-sectional research has indicated that sarcopenia cannot fully account for the loss of strength and power with ageing (Delmonico et al., 2009; Goodpaster et al., 2001; Hughes et al., 2001; Petrella et al., 2005; Visser et al., 2000), which instead is thought to be related to an impaired contractile velocity (Petrella et al., 2005), an increase in non-contractile tissue (i.e. infiltration of fat into the muscle; Goodpaster et al., 2001; Runge et al., 2004), decreases in nerve conduction velocity (Metter et al., 1998) and fascicle length (Narici et al., 2003), impaired ATPase activity in a given fibre (Larsson et al., 1997), E-C 'uncoupling' (Payne & Delbono, 2004), a decrease in the number of motor units (Brown, 1972; Campbell et al., 1973) and an impaired ability to activate the surviving motor units (Kamen et al., 1995).

Losses in power with ageing are well established (Candow & Chilibeck, 2005; Izquierdo et al., 1999; Newton et al., 2002; Petrella et al., 2005; Petrella et al., 2007; Raj, 2010; Runge et al., 2004; Skelton et al., 1994). For example, during elbow and knee flexion and extensions, older males (~66 years) produced lower power than their younger (~23 years) counterparts at both slow (1.05 $rad \cdot s^{-1}$) and fast (3.14 $rad \cdot s^{-1}$) movement velocities (Candow & Chilibeck, 2005). Though, because Candow and Chilibeck's (2005) group was healthy, but not physically active, the older group might have been more susceptible to these age-associated decrements. When activity levels, but not resistance training, were matched between young (~23 years) and old (~62 years) males, these age-associated differences were found to remain (Nogueira et al., 2013). During more complex multi-jointed tasks (i.e. bench throws and countermovement jumps) performed by habitually active males, Izquierdo et al. (1999) also observed an impaired upper- and lower-body power production in older (60 to 74-year-olds) compared to middle-aged (35 to 46-year-olds) participants.

Regarding the factors which might contribute to power, studies by Bean and colleagues (Bean et al., 2002; Bean et al., 2003) determined that leg strength was highly correlated with lower-body power in ~73 year-olds. That similarly high correlations have also been documented in young populations (Baker & Nance, 1999; Carlock et al., 2004; Stone et al., 2003), highlights the importance of strength to power production regardless of age (Baker, 2001). However, despite power being a product of force and velocity, and Petrella and colleagues (2005) suggesting that age-related decreases in power are caused by impaired contractile velocity, no study has established the nature of the relationship between power and velocity specifically in middle-aged males. Such information would elucidate some of the variables (i.e. strength and velocity) that contribute to power in middle-aged males and thus have practical implications for future training models.

The literature on ageing and power is typically health-related and incorporates cohorts of people aged 60 years and above, many of whom neither play sport nor resistance train. As such, the differences in power between the young and old populations are unsurprising. From the perspective of strength and conditioning practitioners who coach athletes of all ages, it would be helpful to know if these findings extend to middle-aged males who habitually resistance train and play sport. If these age-associated reductions are still present in such people, it could be problematic for those who want to be competitive, given the importance of power for many sporting tasks in general, and playing standard in particular (Baker, 2001a; Baker, 2001b; Cronin & Hansen, 2005; Loturco et al., 1993; Lyttle et al., 1996; Sleivert & Taingahie, 2004). However, as no study has investigated this, the purpose of this study is to provide a detailed analysis of the load-velocity and load–power relationships during multi-jointed exercise in young (18 to 25 years) and middle-aged (35 to 55 years)

males who regularly resistance-train. A further aim is to determine the relationship of strength and velocity to peak power in these age groups to elucidate the factors which contribute to power in resistance trained middle-aged males.

## 4.2 Methods

### *4.2.1 Participants*

Twenty young (range: 19 to 25 years) and 20 middle-aged (range: 35 to 54 years) males, with a minimum of two years of resistance training, were recruited via convenience sampling from the University population and local gymnasia for this study. Thirty-five years was selected as the lower boundary for the middle-aged group because it is the entry age for 'Masters' athletes (see British Masters Athletic Federation and World Masters Athletics). As age-related studies typically use older groups (60 years and over), 55 was selected as the upper-limit for the middle-aged group. A sample size of 38 (19 per group) was estimated using G*power 3.1 (Faul, Erdfelder, Buchner & Lang, 2009) based upon an ES, alpha error probability and power of 1.1 (as observed between groups by Aoki & Demura, 2011, for handgrip power at 50% MVC), 0.05 and 0.95, respectively. Participants completed a pre-test health questionnaire and provided written consent for the study, which was approved by the Ethics Committee of the Faculty of Life Sciences at the University of Chester. Participants were instructed not to consume any ergogenic supplements (for example, caffeine) on the day of testing and to refrain from strenuous exercise in the three hours before testing.

*4.2.2 Study design*

This study comprised a mixed factorial design in which two groups of participants attended the laboratory on two occasions and provided repeated measures during three resistance exercises. On the first visit, anthropometric measurements of stature, body mass and body composition were recorded, followed by assessments of maximal strength on bench press (BP), squat (SQT) and bent-over-row (BOR), and habituation to the measures of barbell velocity and power. Participants were considered 'habituated' when they could complete three consecutive repetitions that produced power within ±10% of each other (Batterham & George, 2003). Participants returned to the laboratory 48 hours later to complete three repetitions of BP, SQT and BOR at loads corresponding to 20 to 80% 1RM (at 10% 1RM increments) in a randomised order for both exercise and load.

*4.2.3 Procedures*

*4.2.3.1 Biometric measurements*

Body mass and stature were determined using digital scales (Seca 813, Hamburg, Germany) and a wall-mounted stadiometer (Harpenden, Holtain, Crymych, Dyfed, UK), respectively. Body density was estimated via skinfold measurements (Harpenden, British Indicators, Burgess Hill, UK) taken at the triceps, abdominal, suprailliac and mid-thigh sites and incorporated into the Jackson and Pollock (1985) equation:

$$\text{Body density} = (0.29288 \times \sum \text{skinfolds}) - (0.0005 \times \sum \text{skinfold}^2) + (0.15845 \times \text{age}) - 5.76377$$

Body fat percentage was derived from Db using the Siri (1956) equation:

$$\text{Body fat percentage} = [(4.95/ \text{body density}) - 4.5] \times 100$$

From this quantities (kg) of fat-mass (FM) and fat-free mass (FFM) were derived to determine any age-associated differences in body composition between the groups.

*4.2.3.2 Training history*

Participants completed a questionnaire that required them to detail how many years they had participated in regular resistance training, their weekly training frequency and session duration, and the main reason for their training. This information was collected to help elucidate any age-related differences that might be observed.

*4.2.3.3 Strength testing*

Participants' maximum strengths on the BP and BOR exercises were assessed directly using a standardised 1RM protocol (Stock et al., 2011). For safety reasons, 1RM for SQT exercise was predicted via a 5RM protocol as outlined by Reynolds, Gordon and Robergs (2006) using the equation:

$$1RM\ (kg) = 1.0970 \times (5RM\ weight\ [kg]) + 14.2546$$

The above equation was reported to yield accurate 1RM predictions ($R^2$ = 0.988, standard error of estimate = 13.51 kg; Reynolds et al., 2006).

*4.2.3.4 Assessment of peak power and velocity*

Peak power and velocity were assessed during the three exercises at loads corresponding to 20, 30, 40, 50, 60, 70 and 80% 1RM. Loads were applied in a randomised order with measurements of peak velocity and power recorded using a FitroDyne rotary encoder (Fitronic, Bratislava, Slovakia) attached directly under a Smith machine bar (Smith Machine standard, Perform Better, Leicester, UK) by its

nylon cable. The FitroDyne measures rate of displacement and thus assumes that the nylon cord is moving in a vertical plane. Any deviation from this plane could increase measurement error. As such the Smith machine was employed as it restricts the movement of the nylon cord to the vertical plane only. The FitroDyne is deemed to provide a reliable marker of moderate changes in peak power and velocity during the selected exercises (Chapter 3).

For the BP exercise, the participant held the bar with a prone grip and lowered it to his chest, before maximally pushing it until full elbow extension. For the SQT exercise, with the bar positioned across their shoulders participants descended until their hips were below the knee joint and then ascended as rapidly as possible until their knees were at full extension. A bench was employed to ensure that each participant attained the same depth and range of motion on each repetition. During BOR exercise the participant commenced in a bent-over position, before pulling the bar maximally until the elbows reached full flexion. Three repetitions of each exercise were performed at each load with self-selected rest intervals that were capped at 90 s, but ranged from 30 to 90 s (Jennings et al., 2005). Rest times were self-selected as lighter loads did not require the same recovery time. Peak velocity values were recorded from which peak power (W) was calculated (load x 980 $cm \cdot s^{-1}$ x velocity). For each exercise the load that represented maximal power was deemed the optimal load. Total peak power was calculated as the sum of peak power values of all seven loads.

### *4.2.4 Statistical analysis*

Comparisons of categorical training history variables (i.e. weekly training frequency, session duration and reason for training) by group were made using a Chi-squared

($\chi^2$) test of association. Biometric and training years variables were analysed using an independent *t* test. Peak values of velocity and power were averaged for the three repetitions at each load and their distributions checked for normality and homogeneity of variance using the Shapiro-Wilk and Levene statistics, respectively. Both assumptions were found to be satisfied ($P > 0.05$). Accordingly, a two-way (load x group) analysis of variance (ANOVA) was used to assess the variation of scores. If the assumption of sphericity was not met the Greenhouse-Geisser correction was used. Partial correlation coefficients were calculated to provide an estimation of the contribution of maximal velocity (at 20% 1RM) and 1RM to power at the load that optimised power (50, 80 and 80% 1RM for BP, SQT and BOR, respectively). For all partial correlations the variables not being analysed were controlled for (e.g. the relationship between maximal velocity and power, controlling for 1RM). Effects sizes (ES) for velocity and power output were determined using Cohen's *d,* calculated as the difference between the means divided by the pooled standard deviation of the two groups (Hopkins, 2006). The practical significance of the findings was quantified as: trivial <0.2, small 0.2-0.59, moderate 0.6-1.19, large 1.2-1.99, and very large >2.0 (Hopkins, 2006). All data analyses were performed in SPSS (Version 21, IBM SPSS Inc., Chicago, Il.)

## 4.3 Results

### *4.3.1 Biometric measures and training history*

Mean body mass was not different between groups ($P > 0.05$, ES = 0.31), though FFM ($t_{(38)} = 2.6$, ES = 0.85 ) and FM ($t_{(37.9)} = 3.0$, ES = 0.96) were ($P < 0.05$). A group x exercise type interaction was noted for 1RM ($F_{(1.4, 50.8)} = 6.4$, $P < 0.05$), with the middle-

aged group being weaker ($P < 0.05$) in each exercise, particularly the SQT (-27.7%), than the young group (Table 4.1).

**Table 4.1** Biometric characteristics of the young and middle-aged groups

| | *Young (n = 20)* | *Middle-aged (n = 20)* | *Effect size* |
|---|---|---|---|
| Age (y) | 21.0 ± 1.6 | 42.6 ± 6.7* | 4.55 (very large) |
| Stature (m) | 1.8 ± 0.1 | 1.8 ± 0.1 | 0.00 (none) |
| Mass (kg) | 85.9 ± 12.8 | 82.3 ± 11.2 | 0.31 (small) |
| Fat-free mass (kg) | 77.0 ± 10.7 | 69.0 ± 8.6* | 0.85 (moderate) |
| Fat mass (kg) | 10.1 ± 4.5 | 14.3 ± 4.5* | 0.96 (moderate) |
| Body fat percentage | 11.6 ± 4.0 | 17.2 ± 4.1* | 1.42 (large) |
| Bench press 1RM (kg) | 104.3 ± 17.2 | 85.1 ± 16.2* | 1.18 (moderate) |
| Squat 1RM (kg) | 137.5 ± 26.3 | 99.4 ± 28.6* | 1.42 (large) |
| Bent-over-row 1RM (kg) | 96.5 ± 14.7 | 83.9 ± 12.3* | 0.95 (moderate) |

*significantly different to Young group ($P < 0.05$)

The middle-aged group had regularly resistance trained longer than the young group ($t_{(19.4)} = 4.8$, $P < 0.05$, Table 4.2), but there was a trend for the middle-aged group to conduct their training with a lower weekly frequency ($\chi^2 = 8.1$, $P < 0.05$) and shorter session duration ($\chi^2 = 18.9$, $P < 0.05$). Additionally, the middle-aged group typically resistance trained to improve strength and health, whilst the young group trained solely for hypertrophy and strength gains ($\chi^2 = 13.9$, $P < 0.05$).

**Table 4.2** Training characteristics of the young and middle-aged groups

| | *Young* | *Middle-aged* |
|---|---|---|
| **Years of resistance training** (mean ± SD) | 4.5 ± 1.1 | 16.9 ± 11.4* |
| **Weekly frequency** ** | | |
| *1 to 2* | 1 (5%) | 7 (35%) |
| *3 to 4* | 13 (65%) | 12 (60%) |
| *5+* | 6 (30%) | 1 (5%) |
| **Session duration** ** | | |
| *0 to 30 minutes* | 0 (0%) | 3 (15%) |
| *31 to 60 minutes* | 6 (30%) | 16 (80%) |
| *61 to 90 minutes* | 12 (60%) | 1 (5%) |
| *90+ minutes* | 2 (10%) | 0 (5%) |
| **Reason for resistance training** ** | | |
| *Strength* | 11 (55%) | 7 (35%) |
| *Hypertrophy* | 9 (45%) | 3 (15%) |
| *Fat loss* | 0 (0%) | 0 (0%) |
| *Health* | 0 (0%) | 10 (50%) |

*significantly different to Young group ($P < 0.05$). **significantly associated ($P < 0.05$) Brackets denote percentage of responses in each category.

*4.3.2 Peak velocity*

For BP, the group ($F_{(1, 38)} = 10.5$, $P < 0.05$, ES = 1.7 to 0.0) and load ($F_{(2.1, 79.4)} = 943.4$ $P < 0.05$) effects reflected mean values that were greater for the young group, and decreasing as load increased; Figure 4.1A. The load x group interaction ($F_{(2.1, 79.4)} = 14.1$, $P < 0.05$) revealed narrowing group differences as intensity increased, whereby effects were small and trivial from 60% 1RM onwards. Similar patterns of variability to BP were observed for both the SQT and BOR exercises, albeit the group differences were consistently greater across all loads, and remained moderate and large even at the higher intensities (Figures 4.1B & 4.1C)

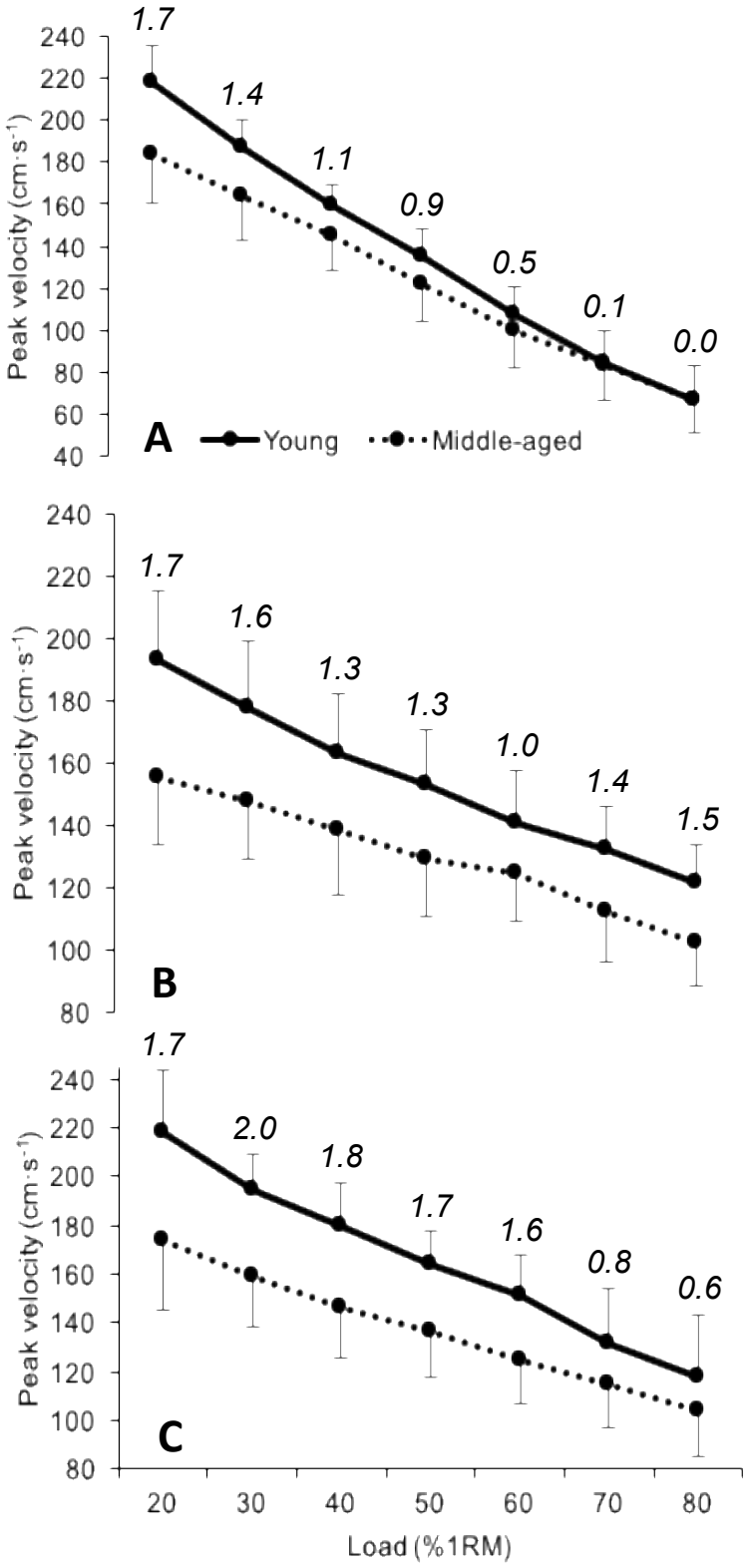


**Figure 4.1** Load-peak velocity relationships in young and middle-aged males during bench press (A), squat (B) and bent-over-row (C) exercises. (Values in *italics* indicate ES).

*4.3.3 Peak power*

As expected from the velocity data, the group effect on BP peak power ($F_{(1, 38)}$ = 31.4, $P$ < 0.05; Figure 4.3A) was significant, with the young group producing higher values than the middle-aged group at all loads (ES = 1.1 to 2.0). Likewise, the effect of load was significant ($F_{(1.8, 65.6)}$ = 943.5, $P$ < 0.05), with peak power being highest (optimised) at 50% 1RM in each group, though the interaction effect was not ($P$ < 0.05). The patterns of peak power values for the SQT and BOR exercises were similar to each other, reinforcing the aforementioned group and load effects seen for bench press. However, distinctive for these two exercises was the optimised values occurring at the highest loads (80% 1RM), and significant ($P$ < 0.05) load x group interactions reflecting (generally) group differences widening with increasing intensities (Figures 4.3B and 4.3C).

Total peak power was significantly higher in the young group compared to the middle-aged group during BP (3996.7 ± 707.3 and 2969.3 ± 623.6 W, respectively, $t_{(38)}$ = 3.4, $P$ < 0.05), SQT (6597.8 ± 1452.5 and 4197.5 ± 1090.4 W, respectively, $t_{(36)}$ = 5.9, $P$ < 0.05) and BOR (4798.3 ± 1031.4 and 3493.6 ± 745.3 W, respectively, $t_{(38)}$ = 4.9, $P$ < 0.05). Moreover, an interaction effect (group x exercise) was observed for total power with the magnitude of the differences being greater between the groups for SQT and BOR (ES = 2.0 and 1.6, respectively) compared to BP (ES = 1.1; Figure 4.2).

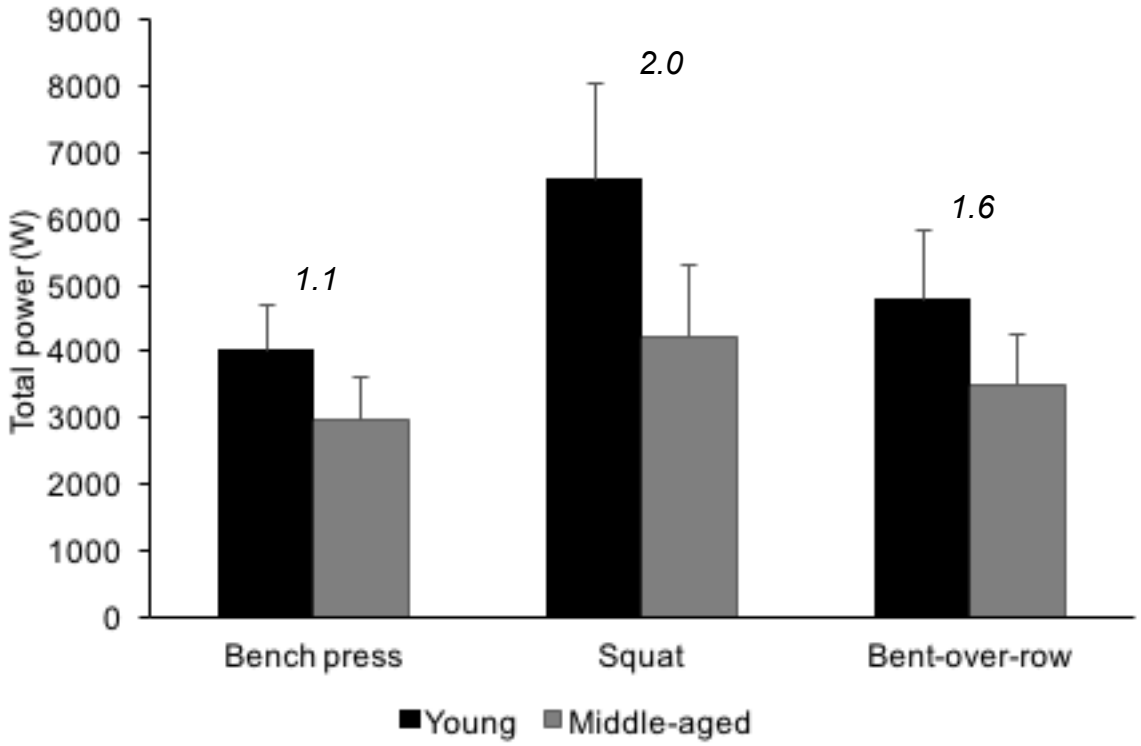


**Figure 4.2** Total peak power in young and middle-aged males during bench press, squat and bent-over-row exercises. (Values in *italics* indicate ES).

*4.3.4 Partial correlations*

For BP exercise in the young group only, 1RM was significantly correlated (Table 4.3) with optimal power output ($r$ = .863, $P$ < 0.05) when controlling for velocity, whereas correlations for both velocity and 1RM were strong and significant in the middle-aged group ($r$ = .846 and .782, respectively, $P$ < 0.05). Both velocity and 1RM were moderately correlated with optimal power output during SQT exercise in the middle-aged group ($P$ < 0.05), while in the young group these correlations were moderate and strong, respectively, during SQT exercise ($P$ < 0.05). During BOR, optimal power output in the young group was only related to 1RM ($r$ = .725, $P$ < 0.05) whilst both velocity and 1RM was strongly correlated to power output in the middle-aged group ($P$ < 0.05).

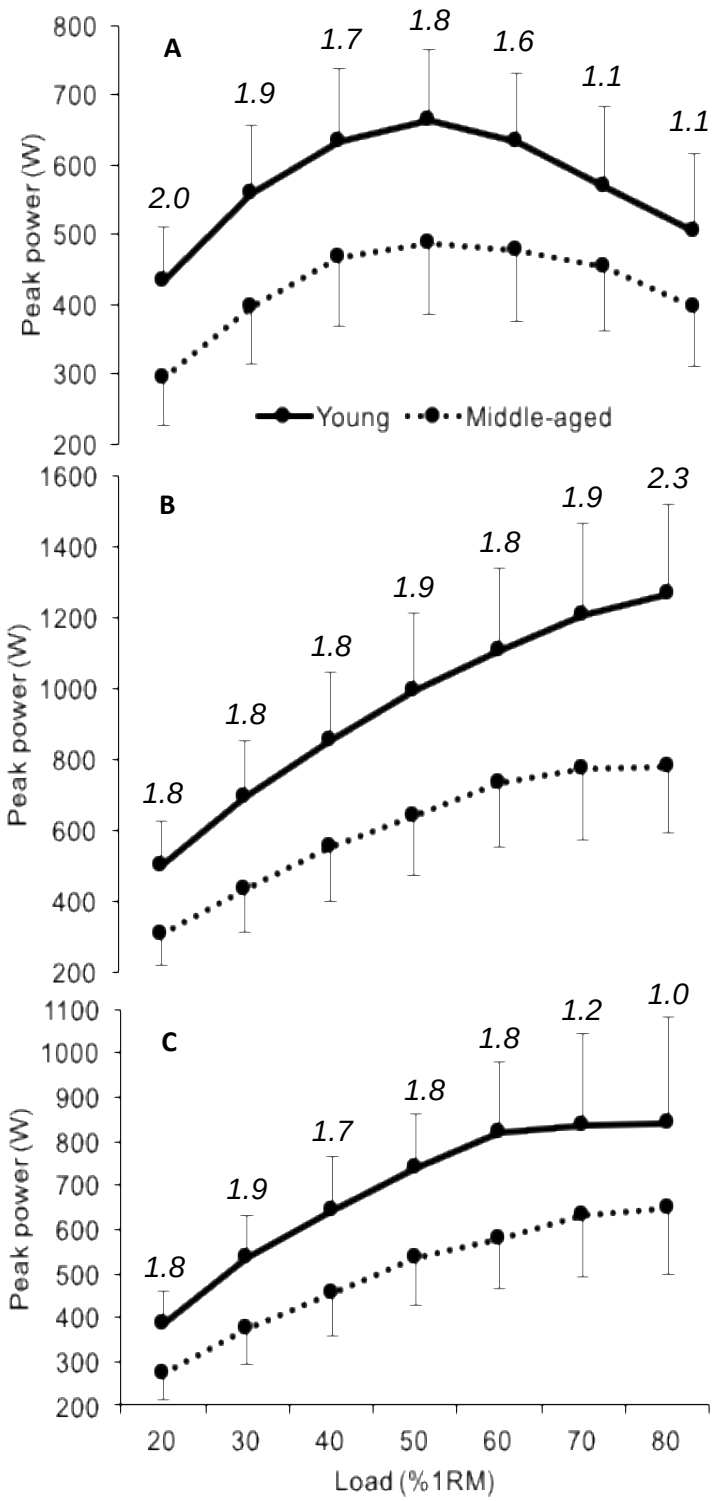


**Figure 4.3** Load-peak power relationships in young and middle-aged males during bench press (A), squat (B) and bent-over-row (C). (Values in *italics* indicate ES)

**Table 4.3** Partial correlations for velocity (controlling for 1RM) and 1RM (controlling for velocity) with optimal power.

| | **Bench press** | | **Squat** | | **Bent-over-row** | |
|---|---|---|---|---|---|---|
| Group | Velocity | 1RM | Velocity | 1RM | Velocity | 1RM |
| *Young* | .404 | .863* | .653* | .877* | .379 | .725* |
| *Middle-aged* | .782* | .846* | .591* | .614* | .753* | .711* |

*significant correlation ($P < 0.05$)

## 4.4 Discussion

This is the first study to provide a comprehensive cross-sectional analysis of the load-velocity and load–power relationships in young and middle-aged athletes who regularly resistance-train. Importantly, these findings indicate that middle-aged, resistance trained males are unable to achieve the high velocities and power outputs during multi-jointed resistance exercises produced by younger counterparts.

Despite between-group similarities in body mass, the middle-aged athletes had a lower FFM and a higher FM than the younger athletes. These age-associated differences in body composition are expected and well documented (Candow & Chilibeck, 2005; Petrella et al., 2005; Runge et al., 2004). Differences between age groups are probably explained by more IMAT (Boettcher et al., 2009; Forsberg et al., 1991; Goodpaster et al., 2001; Jubrias et al., 1997; Zoico et al., 2010) present in the middle-aged group, and their training history, typically incorporating shorter and less frequent resistance training sessions. This idea of a lower training volume would reflect the documented age-associated increases in time spent involved in other things, such as working (Weir et al., 2002) and family-related responsibilities (Tanaka & Seals, 2008). Moreover, the middle-aged males chose resistance training to maintain health and strength, whereas the younger males tended to train this way to increase strength and hypertrophy. Such differences in training goal orientation between groups have

also been noted among Masters athletes who reported training for ‘general health’ and ‘weight concern’ (Ogles & Masters, 2000).

As expected, the middle-aged group was weaker than the young group for all three exercises. These differences in muscle strength are similar to those noted previously for both the upper (Anton et al., 2004; Candow & Chilibeck, 2005; Frontera et al., 1991; Frontera et al., 2000; Izquierdo et al., 1999) and lower-body (Anton et al., 2004; Candow & Chilibeck, 2005; Frontera et al., 1991; Frontera et al., 2000; Frontera et al., 2008; Izquierdo et al., 1999; Newton et al., 2002; Runge et al., 2004; Winegard et al., 1996), and are likely explained by age-associated differences in ‘muscle quality’ (Frontera et al., 2000) and motor unit number (Brown et al., 1972; Campbell et al., 1973) and activation (Kamen et al., 1995). The calculation of ‘muscle quality’ (i.e. strength or peak power relative to muscle mass) might help to elucidate some of the differences between groups. However, the present study’s use of skinfolds to allow such a calculation is too non-specific (i.e. it includes muscle, bone, water, connective tissue and internal organs) to allow such a calculation. The aforementioned training focus of the two groups is again pertinent, with the younger group’s specific concern being that of improving strength (and hypertrophy) and, unlike the middle-aged group, not health. As adaptations to resistance training appear to be specific to the type of training regularly performed (Buckner, Dankel, Mattocks & Leonneke, 2016), it is unlikely that the middle-aged group optimised strength gains from their health-related training.

For all exercises and loads except 60 to 80% 1RM BP, the young group produced higher barbell velocities than the middle-aged group. Although only the studies of Labarque et al. (2002) and Valour et al. (2003) generated statistics in the same manner as the current study (ES = 0.07 and 0.98, respectively, for maximal

velocity elbow flexion), statistical differences between age groups have been documented in upper-body pushing, (Izqueirdo et al., 1999), lower-body (Allison et al., 2013; Krivackas et al., 2001; Larsson et al., 1997; Petrella et al., 2005; Ochala et al., 2007; Thom et al., 2005; Thom et al., 2007; Yu et al., 2007) and upper-body pulling exercises (Monemi et al., 1999, Monemi et al., 1998; Toji & Koneko, 2007). Although not measured in this study, it is plausible that the age-associated differences in fasiscle length (Narici et al., 2003; Morse et al., 2005; Thom et al., 2007), reduced ATPase activity (Larsson et al., 1997), type 2a fibre atrophy (Lexell et al., 1988), and changes in contractile properties (i.e. increased slow myosin heavy chain content; Valour et al., 2003) which contribute to maximal velocity contractions, contributed to the poorer performance of the middle-age group. Indeed, the small to non-effects at 60 to 80% 1RM BP might be explained by the low movement velocities exhibited by both groups. That is, the mechanisms noted above might not be sufficient to induce these age-associated differences when movement velocities are very low. That there were differences in barbell velocities between the groups during SQT and BOR at 60 to 80% 1RM, when the average barbell velocities were higher (111.9 to 132.6 and 110.9 and 137.4 $cm \cdot s^{-1}$ for SQT and BOR, respectively) than BP (66.6 to 103.6 $cm \cdot s^{-1}$), would support this notion.

Peak power during BP, SQT and BOR was superior in the young athletes, with moderate to very large differences between groups. Whilst Allison et al. (2013) and Jozsi et al. (1999) have noted very large effects (ES = 1.86) for leg press, and small to moderate effects (0.23 to 0.95) for seated arm pull between young and old groups, many more studies have observed statistical differences between young and older groups during upper-body pushing (Anton et al., 2004; Candow & Chilibeck, 2005; Izquierdo et al., 1999; Ojanen et al., 2007), lower-body (Anton et al., 2004; Candow &

Chilibeck, 2005; Izqueirdo et al., 1999; McNeil et al., 2007; Newton et al., 2002; Petrella et al., 2005; Thom et al., 2005; Thom et al., 2007) and upper-body pulling exercises (Aoki et al., 2011; Candow & Chilibeck, 2005; Toji & Kaneko, 2007). As for velocity (above) such discrepancies in power can be explained in terms of reduced calcium release (Delbano et al., 1995) and muscle morphology, that is, reduced fascicle length, and subsequent force production (Blazevich & Sharp, 2005; Narici et al., 2003; Morse et al., 2005; Thom et al., 2007), and an impaired motor unit activation and number (Brown, 1972; Campbell et al., 1973; Kamen, 1995). That the effects between age groups in the current study were greater for power than velocity (for all exercises) suggests that the lower strength of the middle-age athletes more likely accounted for their lower power outputs than barbell velocity.

The greater between-group differences observed in lower-body total power and strength than upper-body sits well with prior literature reporting site-specific strength and power disparities (Candow & Chilibeck, 2005; Frontera et al., 2000; Lynch et al., 1999; Noguiera et al., 2013). Though the phenomenon is not particularly well understood, it has been suggested that during daily living lower-body movements are supplemented by upper-body contributions (e.g. using the upper-body to rise from a chair; Macaluso & DeVito, 2004) and the lower-body undergoing more severe changes in muscle contractile units (e.g. decreases in the specific tension of type 1 and 2 fibres; Larsson et al., 1997) and connective tissues (e.g. increases in fat and connective tissue; Lynch et al., 1999; Overend et al., 1992). Practically, these site-specific differences in strength and power suggest that middle-aged athletes may need to undertake methods to offset such differences.

For BP, strength was strongly correlated ($r > 0.84$) with power output in both young and middle-aged groups, reaffirming the work of others (Baker & Nance, 1999;

Baker, 2001; Marques et al., 2007). However, unlike previous research, velocity was strongly correlated with peak power only in the middle-aged group. This suggests that higher power production in upper-body pushing exercise in middle-aged males is achieved from greater strength and higher barbell velocity, whereas in the young group higher power is achieved via greater strength only. For SQT exercise, strength was highly ($r$ = 0.877) and moderately ($r$ = 0.614) correlated with peak power in the young and middle-aged athletes, respectively, and reaffirms previous findings among young (Baker & Nance, 1999; Carlock et al., 2004; Stone et al., 2003) and older populations (Bean et al., 2002; Bean et al., 2003). Peak power illustrated a moderate relationship with velocity in both groups during SQT exercise. It appears that the middle-aged group is equally reliant on strength and velocity when producing power during SQT exercise. Thus, it would be appropriate for both young and middle-aged males to focus increasing on both strength and barbell velocity to increase their power. For BOR, strength formed a strong relationship with peak power in both the young and middle-aged groups ($r$ = 0.725 and 0.711, respectively). The reason for the non-significant correlations between velocity and power in the young group, but strong correlations in the middle-aged group, is unclear but does indicate that to increase power middle-aged athletes require improvements in both strength and velocity. Collectively, this correlation data supports the notion that to produce high power, individuals must first be relatively strong (Cormie, McGuigan & Newton, 2011; Haff & Nimphius, 2012). A variety of studies in older populations (~66 to 74 year olds) have noted that high-velocity power training to be more effective than low-velocity/high-strength training (Bottaro, Machado, Nogueira & Veloso, 2007; Ramirez-Campillo et al., 2014; Sayers & Gibson, 2010; Sayers & Gibson, 2014). Thus, middle-aged athletes would benefit

from adopting a training approach which maximises both strength and velocity adaptations.

### 4.5 Conclusion

Though a cross-sectional design, this study provides a comprehensive analysis of the load-velocity and load–power relationships exhibited during three popular exercises among resistance trained young and middle-aged males. These data indicate that in comparison to younger athletes, middle-aged athletes were unable to achieve high barbell velocities at low external resistances. Moreover, power during BP, SQT and BOR was particularly diminished in the middle-aged group. These impairments in velocity and power might explain some of the age-associated decreases in sporting performance previously reported in middle-aged athletes. Given the strong relationships between strength and velocity with power in the middle-aged group, such athletes should undertake specific training methods to improve both components.

# 5. Internal loads, but not external loads and the fatigue response, are similar in young and middle-aged resistance trained males during high volume squatting exercise

## 5.1 Introduction

Longitudinal resistance training induces muscle hypertrophy and increases in strength and power that are independent of age (Newton et al., 2002). While such morphological adaptations have been noted in younger athletes (Newton et al., 2002; Schoenfeld, Peterson, Ogborn, Contreras & Sonmez, 2015), they have also been observed in older populations (Bottaro, Machado, Nogueira & Veloso, 2007; Kongsgaard, Backer, Jorgensen, Kjaer & Beyer, 2004; Kosek, Kim, Petrella, Cross & Bamman, 2006; Newton et al., 2002; Roth et al., 1999; Sayers & Gibson, 2010; Sayers & Gibson, 2014), for whom natural age-associated losses in muscle mass (sarcopenia; Narici et al., 2004; Welle, 2002) and strength and power (dynapenia; Chapter 4) are expected. For the growing number of 'middle-aged' athletes (i.e. those 35 to 55 years; Chapter 4; Tanaka & Seals, 2008), resistance training can off-set or delay the effects of sarcopenia and dynapenia to maintain sporting performance (Baker & Tang, 2010; Pantoja et al., 2016).

To determine the efficacy of an athlete's resistance training a coach must quantify the stress imposed on the athlete (Scott et al., 2016). If the training load is insufficient then adaptation might not occur, whereas excessive or sudden increases in stress might result in injury or poor performance (Foster, 1998; Halson, 2014). As such, practitioners should record markers of internal (i.e. HR, RPE) and external (i.e. velocity, power) load to quantify the training stress. However, because of the numerous factors (e.g. movement velocity, rest times, relative intensity, volume load)

that can invoke a resistance training adaptation there is no consensus regarding the best method to monitor resistance training load (Scott et al., 2016).

There is evidence to indicate that internal load variables might differ between age groups when exercising at the same relative external load. For example, higher absolute HRs (Smolander et al., 1998) and blood lactate concentration (Smilios et al., 2007; Walker et al., 2013), and lower (Allman & Rice, 2003; Justice et al., 2014), higher (Pincivero, 2011) and similar (Pincivero et al., 2010) RPEs have been noted in young (~21 to 28 years) compared to older (~57 to 84 years) males during resistance exercise. These findings are despite observations of no differences in absolute or relative HR (Kawano et al., 2008) or blood lactate concentration and RPE (Manini et al., 2012) at the point of muscular failure between young (~21 to 28 years) and older (~48 to 67 years) males. Furthermore, to the author's knowledge, no study has yet compared the external load between age groups during resistance training exercise despite external load being the primary driver of resistance training adaptations (Schoenfeld, 2010). A limitation of focusing on external or internal load in isolation is that they might not be able to reflect the internal load for a given external load. Therefore, calculating an internal to external load ratio might negate the poor sensitivity and inter-individual variability of individual training load metrics (Akubat, Barrett & Abt, 2014). The use of external load markers in isolation demonstrates a limited relationship with measures of endurance capacity (velocity at lactate threshold, velocity at 4 $mmol \cdot L^{-1}$ and $VO_{2max}$), whereas the external to internal load ratios exhibit moderate to large correlations ($r$ = .41 to .69; Akubat et al., 2014; Malone et al., 2016). These data suggest that the integration of internal and external load might offer a more sensitive measure of overall training load, however the application to resistance type exercise is yet to be explored.

The subsequent fatigue (i.e. inability to maintain the expected force or power output; Edwards, 1981) response to resistance exercise between age groups is unclear (Avin & Frey Law, 2011; Christie, Snook & Kent-Braun, 2011). Two recent meta-analyses concluded that ageing is associated with less fatigue after isometric contractions, but not dynamic contractions, when assessed in terms of force production during MVCs (Avin & Frey Law, 2011; Christie et al., 2011). When velocity and power are used as markers of fatigue, older (~64 to 75 years) males experience greater fatigue than their young (~27 years) males during knee extension (Dalton et al., 2012; Dalton et al., 2015; Petrella et al., 2005), but not during sit-to-stand exercise (Christie et al., 2011; Petrella et al., 2005). It has been suggested that the age-related slowing of the muscle is responsible for the greater fatigue during knee extension exercise (Dalton et al., 2012; Petrella et al., 2005), whilst the group similarity in fatigue during sit-to-stand exercise was attributed to task specificity; both groups would typically perform sit-to-stand tasks but not knee extension movements (Petrella et al., 2005). However, the findings of these studies might not be applicable to the middle-aged male who regularly resistance exercises and plays sports because single-jointed knee extension and sit-to-stand movements are not applicable to the multi-jointed compound movements involved in such activities. A study that quantifies the fatigue response from an ecologically valid resistance training protocol would therefore be particularly beneficial to the resistance trained middle-aged male.

Another plausible explanation for the differences in the fatigue response between age groups might be sought from the internal and external loads experienced during exercise. That is, greater fatigue might be an artefact of a higher internal or external load during exercise of the same relative load. Resistance training protocols with a large amount of work performed are subject to greater decrements in isometric

force (Brandon et al., 2015; Howatson et al., 2016). However, no study has investigated the relationship between internal load and post-exercise decrements in muscle function. Moreover, despite the efforts of two studies (Kawano et al., 2008; Smilios et al., 2007), the age-related research has focused solely on those aged over ~60 years, none of whom were resistance trained. Thus, the stress imposed during resistance exercise in middle-aged males, compared to younger males, is unknown. The findings from a study that quantifies the internal and external load in middle-aged (35 to 55 years) males would be particularly useful for middle-age men who seek to monitor their resistance training. Consequently, the primary aim of this study was to quantify the internal and external loads experienced in lower-limb resistance exercise in young and middle-aged males who regularly resistance train, and to determine the fatigue responses to such exercise. A further aim was to determine the relationship between internal and external load with post-exercise decrements in muscle function.

## 5.2 Methods

### *5.2.1 Participants*

Nine young (21 to 25 years) and nine middle-aged (35 to 54 years) resistance trained males were recruited for this study from the University population, local gymnasia and sports teams using convenience sampling. Thirty-five years was selected as the lower boundary for the middle-aged group because it is the entry age for ‘Masters’ athletes (see British Masters Athletic Federation and World Masters Athletics). As age-related studies typically use older groups (60 years and over), 55 was selected as the upper-limit for the middle-aged group. All participants took part in sport (i.e. team sports, racket sports and endurance type sports) for a minimum of two years (4.1 ± 1.3 and 18.0 ± 5.6 years for the young and middle-aged groups, respectively), and had a

minimum of two years' resistance training experience and regularly used squats as part of their resistance training programmes. Participants completed a pre-test health questionnaire and provided written consent for the study, which was approved by the Ethics Committee of the Faculty of Life Sciences at the University of Chester.

### *5.2.2 Study design*

The study used a mixed factorial design that required attendance at the strength and conditioning laboratory on two separate occasions. Participants were instructed not to consume any ergogenic supplements (for example, caffeine) on each occasion and to refrain from heavy exercise between visits. On the first occasion, they provided biometric data (stature, body mass and skinfold thicknesses for the assessment of body composition), an estimate of back squat 1RM, and were habituated with the measurements of lower-limb peak power, MVC and VA during isometric knee extension. Participants were considered 'habituated' when they could complete three consecutive repetitions that produced peak powers or torque values each within 10% (Batterham & George, 2003). On returning to the laboratory 2-4 days later, they provided measurements of peak power during squats at 20 and 80% 1RM, MVC, VA and blood lactate before and after an exercise bout comprising 10 x 10 squats at 60% 1RM (MacDonald et al., 2014). During the exercise bout, bar velocity and peak power were recorded for each repetition, and HR and RPE were recorded at the end of each set. Session RPE (sRPE) was recorded 15 minutes after the squatting exercise bout. Participants were not provided with any feedback during the study that might have influenced their sRPE.

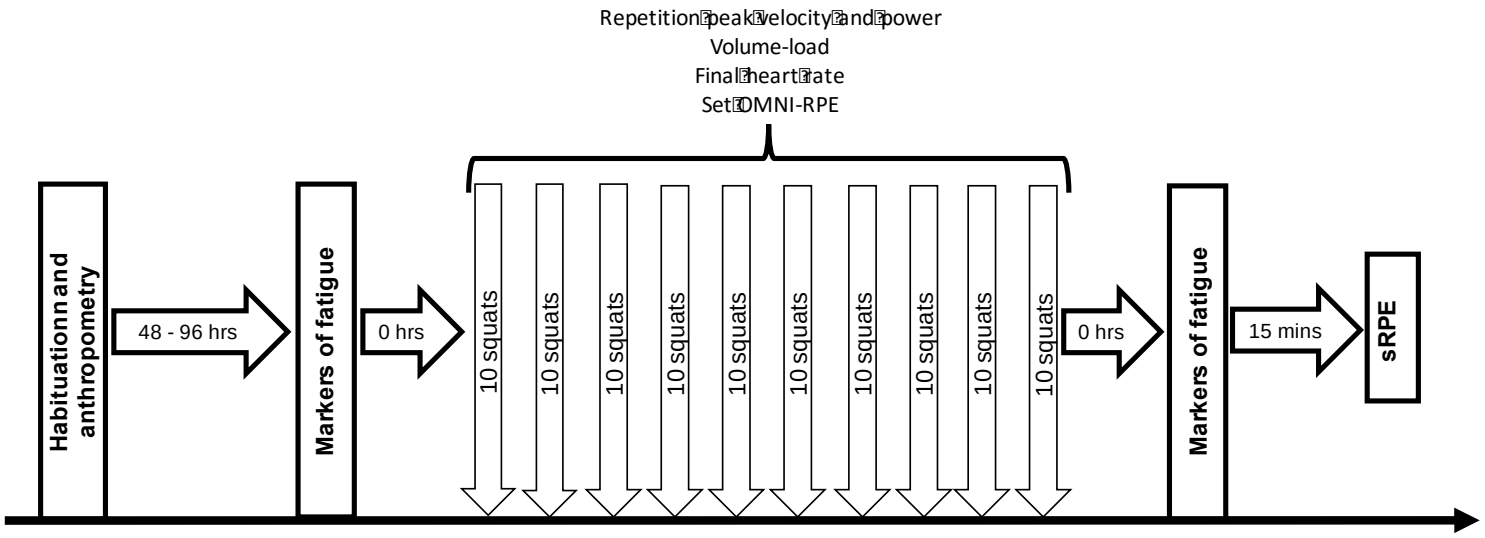


**Figure 5.1** Schematic of study design

*5.2.3 Procedures*

*5.2.3.1 Biometric measures*

Body mass and stature were determined using digital scales (Seca 813, Hamburgm Germany) and a wall-mounted stadiometer (Harpenden, Holtainm Crymych, Dyfed, UK). Body composition was assessed via skinfold thickness measurements (Harpenden, British Indicators, Burgess Hill, UK) taken at the triceps, axilla, abdominal, suprailliac, chest, subscapular, and mid-thigh incorporated into the equation of Jackson and Pollock (1978) for predicting body density. Body fat percentage was derived from the equation (Hayward & Wagner, 2004): body fat percentage = [(5.21/ body density) – 4.78] x 100. From this the quantities (kg) of FM and FFM were also derived.

*5.2.3.2 Maximal strength testing*

To avoid the risk associated with maximal strength testing (Mazur et al., 1993), 1RM for squat exercise was predicted using a 3RM protocol. In brief, participants performed

8-10 repetitions with 50% of their estimated 1RM, followed by 3-5 repetitions at 85% of estimated 1RM. The load was then set at the approximate 3RM and the participants performed three repetitions. The load was progressively increased until the participant could no longer perform a complete repetition. The final load lifted was used with the following equation (Wathan, 1994) to estimate 1RM squat load:

$$1RM = (100 \times \text{load lifted})/(48.8 + (53.8 \times 2.71828^{-0.075 \times \text{repetitions}}))$$

The above equation has been reported to yield accurate 1RM predictions ($r$ = 0.969, 0.02% different from direct 1RM; LeSuer et al., 1997).

*5.2.3.3 Assessment of peak power during back squat*

Peak power was assessed at loads corresponding to 20 and 80% 1RM during back squat exercise using a rotary encoder (FitroDyne, Fitronic, Bratislava, Slovakia) attached via a nylon cord directly under a Smith machine bar (Perform Better, Leicester, UK). As the FitroDyne measures rate of displacement and assumes that the nylon cord is moving in a vertical plane, a Smith machine was used to prevent deviation from this plane and decrease measurement error. The FitroDyne has been shown to produce reliable intra-day measures of peak power (CV = 3.9 - 4.9%) at the selected loads (Chapter 3).

With the bar positioned across the shoulders, participants squatted until their hips were below the knee joint and then ascended as rapidly as possible until their knees were at full extension. A bench was employed to ensure that they attained the same depth and range of motion on each repetition. Three repetitions at each load were performed with self-selected rest intervals that ranged from 30 to 90 s (Chapter

3; Jennings et al., 2005). Rest times were self-selected, as lighter loads (20% 1RM) did not require the same recovery time. Peak velocity was recorded from which peak power was calculated as (load x velocity x 980 $cm \cdot s^{-1}$)/100. The load order was randomised for each participant to negate possible ordering effects.

*5.2.3.4 Assessment of maximal voluntary contraction and voluntary activation*

Before undertaking the MVC and VA assessments, participants performed a warm-up comprising five minutes of cycling at 100 W (Lode, Corival, Groningen, Netherlands). A dynamometer (Biodex, Multi-joint system 3, Biodex Medical, New York, USA) was used to measure isometric force of the participant's dominant knee extensors at 80° knee flexion. To prevent extraneous body movements, Velcro straps were applied tightly across the chest and thigh. Participants were provided with strong verbal encouragement and real-time feedback via the PC monitor.

The knee extensors were electrically stimulated (5 s with two 100 Hz single square impulses (doublet); Digitimer, D57, Hertfordshire, UK) using two 5 x 13 cm moistened surface electrodes (Axelgaard Manufacturing Co LTD, Fallbrook, CA); one placed distally over the quadriceps and the other proximally over the upper quadriceps. During optimisation the amplitude of a doublet was progressively increased, starting at 50 amps, until a point where no further increases in intensity resulted in an increase in resting doublet force. Initially a 230 volt electrically evoked doublet (set 20% above the value required to evoke a resting muscle doublet of maximum amplitude) was applied to the resting muscle (resting doublet) at 1 s. The resting doublet was used to elucidate any peripheral alterations that might have occurred as a result of the squatting protocol (MacDonald et al., 2014). Participants then performed a 4 s MVC before a doublet which was applied at the isometric plateau

(superimposed doublet). The MVC was taken as the average force over 50 ms (AcqKnowledge 3 software, Biopac Systems, Massachusetts) before the superimposed doublet was applied. VA was calculated according to the interpolated twitch ratio (Merton, 1954) using the equation;

VA (%) = [1- (size of superimposed doublet/ size of resting doublet)] x 100

A similar procedure has been deemed a reliable method (CV = 3.38%) for assessing VA (Morton et al., 2005).

*5.2.3.5 High volume squat exercise*

The exercise protocol consisted of 10 sets of 10 repetitions of squat exercise at a load corresponding to 60% 1RM with 120 s rest between sets (MacDonald et al., 2014). For each repetition participants descended for 3 s until their hips were below the knee joint and then ascended as rapidly as possible until their knees reached full extension. A bench was employed to standardise the depth of each repetition. The FitroDyne was used to calculate power for each repetition in the manner outlined above. Mean peak velocity and power over the sets was used to determine the relationship between external load during the exercise and alterations in the markers of fatigue. Volume load was calculated as the 60% 1RM load multiplied by 100.

*5.2.3.6 Assessment of heart rate*

HR was recorded at rest and at the end of each set using a chest strap (Polar Electro, Polar Beat, Oy, Finland).

*5.2.3.7 Assessment of perceived exertion*

At the end of each set participants provided a global indication of their perceived exertion using the OMNI-RPE scale (Robertson et al., 2003), which ranges from 0 to 10, 0 indicating ‘extremely easy’ and 10 corresponding to ‘extremely hard’. Previously, participants were provided with detailed instructions on how to rate their exertion. The OMNI-RPE scale is deemed a valid measure of perceived exertion during resistance exercise (Roberston et al., 2003). Additionally, sRPE (Foster et al., 2001) was recorded 15 minutes after the completion of exercise. Participants were asked “How intense was your session?” and ranked their exertion on a 1 to 10 scale, where 1 indicates “really easy” and 10 indicates “maximal”. This method has been deemed a valid (Sweet et al., 2004) and reliable (Day, McGuigan, Brice & Foster, 2004; McGuigan, Egan & Foster, 2004) indicator of resistance exercise intensity.

*5.2.3.8 Assessment of blood lactate concentration*

Blood was obtained before and immediately after the exercise bout from a finger-tip capillary sample and analysed for lactate concentration using a Lactate Pro analyser (Arkray, Kyoto, Japan). The Lactate Pro has been deemed a reliable marker of blood lactate concentrations (CVs = 2.8 to 5.0%; Baldari et al., 2009).

*5.2.3.9 External to internal load ratios*

External load was quantified using mean peak velocity and power over the 10 sets of exercise and total volume load. Internal load was quantified using measures of mean HR and OMNI-RPE. External load was divided by each measurement of internal load to calculate the external to internal load ratio for the exercise protocol (Akubat et al., 2014).

*5.2.4 Statistical analysis*

All data were analysed using the ES with 90% CL (Hopkins et al., 2009). Magnitude-based inferential statistics were used to provide information on the size of the differences, allowing for a more practical and meaningful explanation of the data (Batterham & Hopkins, 2006). Thresholds for the magnitude of the observed change for each variable were determined as the within-participant standard deviation in that variable x 0.2, 0.6 and 1.2 for a small, moderate and large effect, respectively (Cohen, 1988). Threshold probabilities for a meaningful effect based on the 90% CL were: <0.5% most unlikely, 0.5–5% very unlikely, 5–25% unlikely, 25–75% possibly, 75–95% likely, 95–99.5% very likely, >99.5% most likely. Effects with CL across a likely small positive or negative change were classified as unclear (Hopkins et al., 2009). The rate of change of peak velocity and power, HR and OMNI-RPE during exercise was expressed as the slope of the regression line (beta coefficient; Twist & Eston, 2005) of the dependent variables over the ten sets. A post hoc power calculation indicated that a sample size of 12 to 14 was needed to detect the changes in muscle function observed in the current study. All calculations were completed using predesigned spreadsheets (sportsci.org). Data are presented as ES, lower CL and upper CL. Pearson correlations were employed to quantify the association between the markers of internal and external load and the decrements in muscle function after squat exercise. The following scales were used to interpret the magnitude of the correlations: <0.1 trivial, 0.1-0.3 small, 0.31-0.5 moderate, 0.51-0.7 large, 0.71-0.9 very large, >0.9 nearly perfect (Hopkins et al., 2009). Threshold probabilities for a meaningful effect based on the 90% CL were calculated using a predesigned spreadsheet (Hopkins, 2007).

## 5.3 Results

### *5.3.1 Biometric measures and training history*

Age was *most likely* higher in the middle-aged group compared to the young group (Table 5.1) whilst sum of skinfolds and squat 1RM displayed *likely* differences between groups. Differences in FM and body fat percentage between the young and middle-aged groups were *very likely* between groups while mass was *unclear*.

**Table 5.1** Biometric characteristics (mean ± SD) of the young and middle-aged groups. Qualitative descriptor, ES and upper and lower 90% CL are noted in the ES column.

| Characteristic | Young (*n* = 9) | Middle-aged (*n* = 9) | Effect size |
|---|---|---|---|
| Age (y) | 22.3 ± 1.7 | 39.9 ± 6.2 | *Most likely* ↑ 3.70 (2.87, 4.53) |
| Mass (kg) | 82.0 ± 9.0 | 79.1 ± 10.3 | *Unclear* 0.29 (-1.10, 0.52) |
| Fat-free mass (kg) | 71.4 ± 7.9 | 63.9 ± 6.5 | *Very likely* ↓ -1.02 (-1.83, -0.22) |
| Fat-mass (kg) | 10.5 ± 4.5 | 15.2 ± 5.7 | *Likely* ↑ 0.89 (0.09, 1.70) |
| Body fat (%) | 12.8 ± 4.7 | 18.8 ± 5.8 | *Very likely* ↑ 1.13 (0.32, 1.94) |
| Sum of skinfolds (mm) | 82.3 ± 24.6 | 102.4 ± 31.9 | *Likely* ↑ 0.69 (-0.12, 1.50) |
| Squat 1RM (kg) | 130.8 ± 26.8 | 109.3 ± 22.5 | *Likely* ↓ -0.85 (-1.65, -0.04) |

### *5.3.2 Internal load measures*

Differences in HR (Figure 5.2) and OMNI-RPE (Figure 3) were *unclear* between the young and middle-aged groups over the sets. Differences in mean sRPE (7.7 ± 1.2 and 7.8 ± 1.3 for the young and middle-aged groups, respectively) were also *unclear* (ES 0.09, CL -0.72, 0.89). The rate of change for HR over the sets was *unclear* (ES 0.17, CL -0.63, 0.98) between young (*b* = 1.72 ± 0.96) and middle-aged (*b* = 1.91 ±

1.13) groups, as was the beta coefficient ($b$ = 0.36 ± 0.09 and 0.34 ± 0.17, respectively) for OMNI-RPE (ES 0.17, CL -0.98, 0.65).

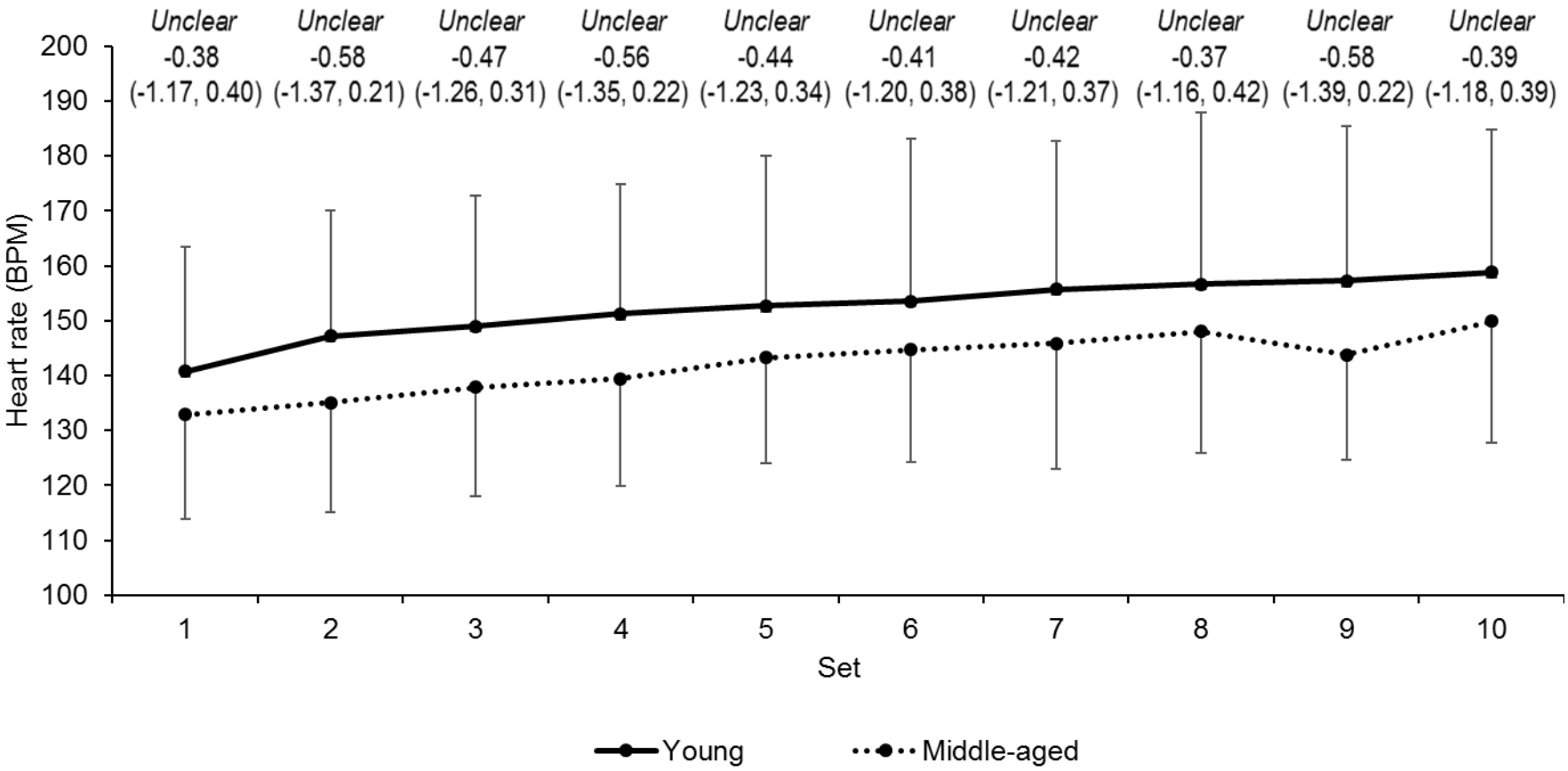


**Figure 5.2** Absolute heart rate scores (mean ± SD) across each set for young and middle-aged groups. Qualitative descriptor, ES and upper and lower 90% CL are noted above.

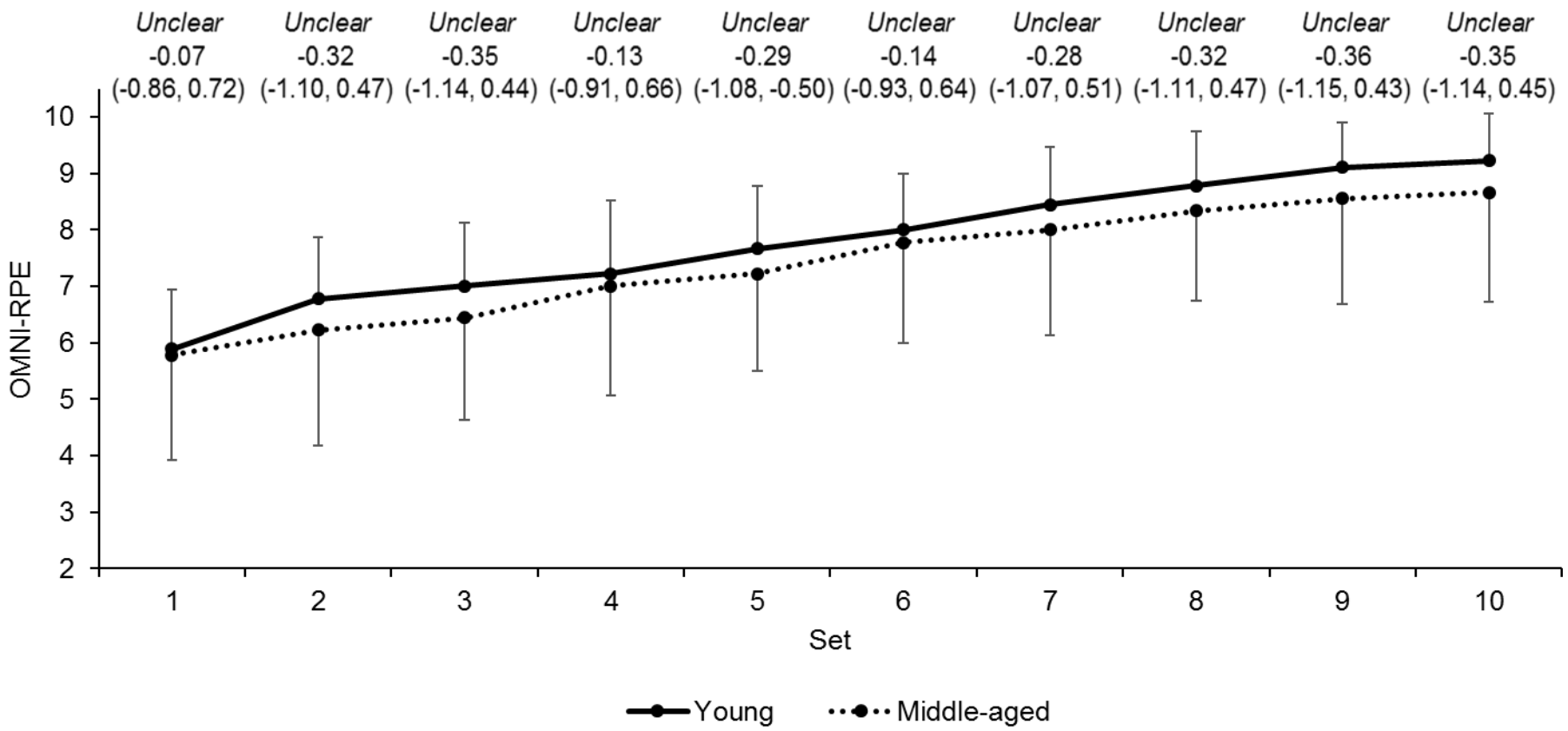


**Figure 5.3** OMNI-RPE scores (mean ± SD) across each set for young and middle-aged groups. Qualitative descriptor, ES and upper and lower 90% CL are noted above.

*5.3.3 External load measures*

Differences in peak velocity over the sets between the young and middle-aged groups were *unclear* (Figure 5.4). Differences in peak power over the sets were *likely moderate* (Figure 5.5) between the groups, except for set 9 where differences were *unclear*. The *unclear* (ES -0.12, CL -0.92, 0.69) differences in mean peak velocity for the young (97.9 ± 24.9 cm/s) and middle-aged (95.2 ± 19.7 cm/s) groups over the sets was accompanied by *likely moderate* differences in mean peak power (ES -0.71, CL -1.53, 0.10; 770.4 ± 278.0 and 603.2 ± 162.6 W for the young and middle-aged groups, respectively). Moreover, there was a *likely moderate* (ES -0.90, CL -1.70, -0.09) higher volume load in young (7898.2 ± 1560.0 kg) group compared to the middle-aged (6556.9 ± 1349.1 kg) group. Differences in mean beta coefficients for velocity and power across the sets were *unclear* (ES 0.31, CL -0.50, 1.11 and ES 0.31, CL -0.51, 1.10, respectively) between young (*b* = -1.7 ± 2.8 and -11.8 ± 20.5, respectively) and middle-aged (*b* = -0.9 ± 2.6 and -5.9 ± 18.2, respectively) groups.

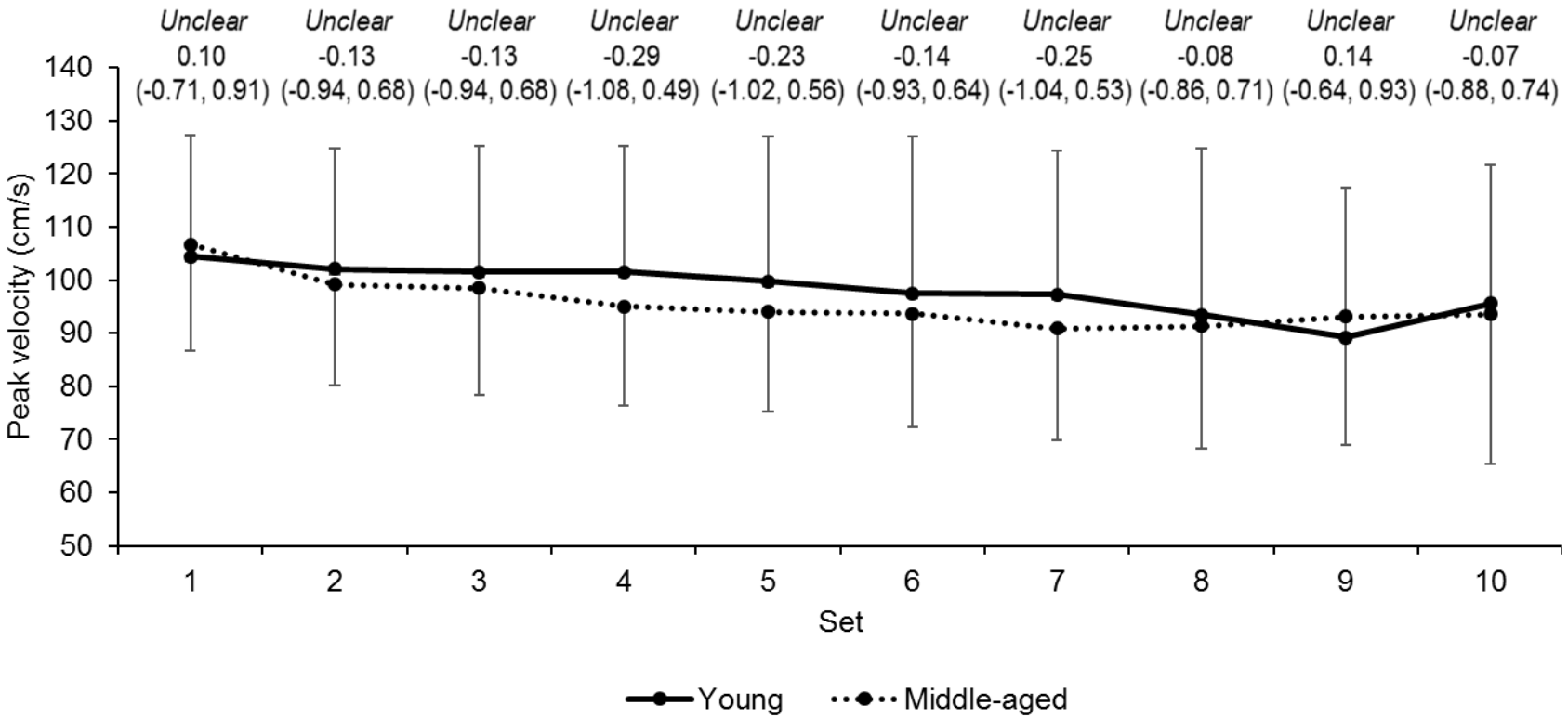


**Figure 5.4** Peak velocity (mean ± SD) across each set in young and middle-aged groups. Qualitative descriptor, ES and upper and lower 90% CL are noted above.

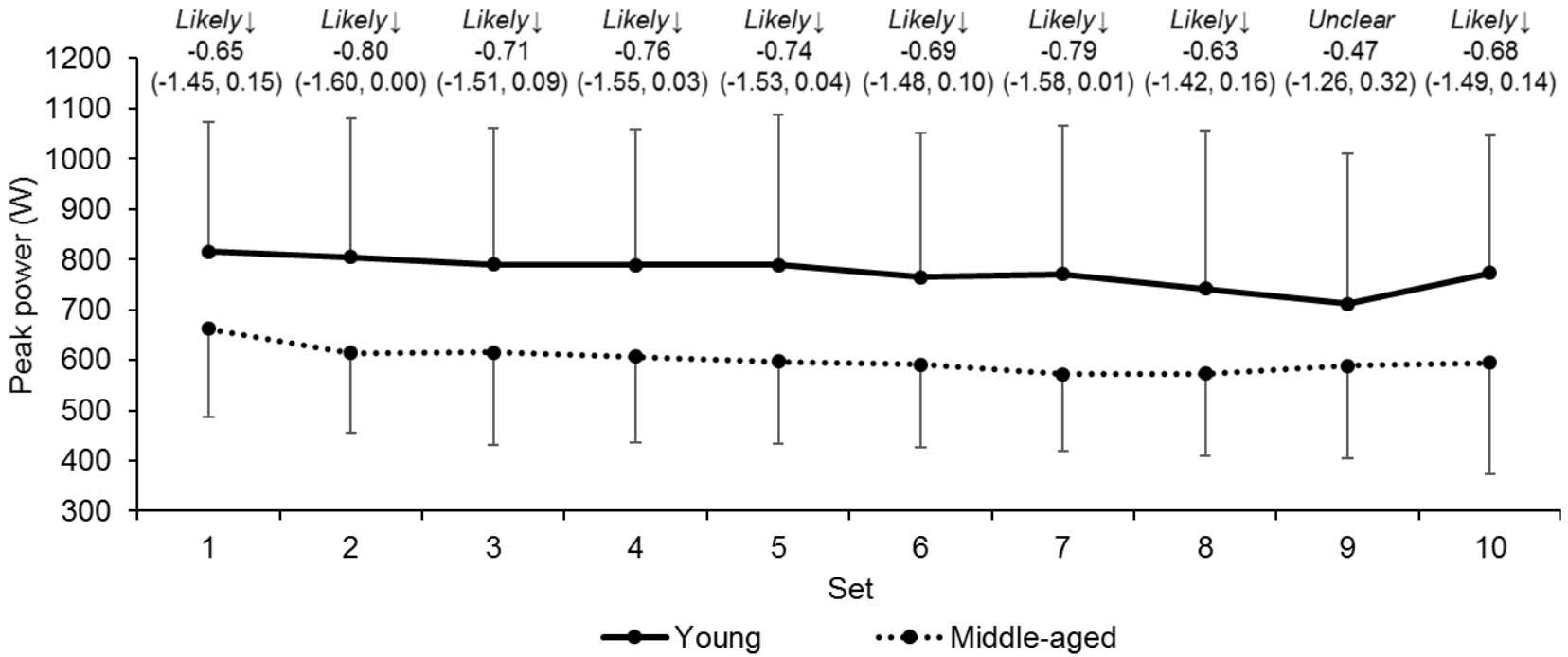


**Figure 5.5** Peak power (mean ± SD) across each set in young and middle-aged groups. Qualitative descriptor, ES and upper and lower 90% CL are noted above.

*5.3.4 External to internal load ratios*

Differences in the external to internal load ratios between the groups were all *unclear* (Table 5.2).

**Table 5.2.** The external to internal load ratio during the exercise protocol in the young and middle-aged groups. Qualitative descriptor, ES and upper and lower 90% CL are noted in the ES column.

| Load Ratio | Young | Middle-aged | Effect size |
|---|---|---|---|
| HR: peak velocity | 0.7 ± 0.2 | 0.7 ± 0.2 | *Unclear* 0.10 (-0.71, 0.90) |
| HR:peak power | 5.2 ± 2.0 | 4.3 ± 1.3 | *Unclear* -0.51 (-1.32, 0.30) |
| HR:volume load | 52.2 ± 11.8 | 47.0 ± 13.0 | *Unclear* -0.41 (-1.22, 0.39) |
| OMNI-RPE: peak velocity | 12.6 ± 3.3 | 13.3 ± 2.7 | *Unclear* 0.21 (-0.60, 1.01) |
| OMNI-RPE: peak power | 99.5 ± 36.6 | 84.8 ± 23.1 | *Unclear* -0.47 (-1.28, 0.34) |
| OMNI-RPE: volume load | 1030.2 ± 244.6 | 968.5 ± 451.2 | *Unclear* -0.14 (-0.95, 0.68) |

*5.3.5 Markers of fatigue after squatting exercise*

At Pre, the *likely moderate* differences in MVC (ES -0.80, CL -1.61, 0.01) and resting doublet force (ES -0.96 CL -1.77, 0.14) between the groups were accompanied by *very likely moderate* differences in 20 (ES -1.03, CL -1.84, -0.22) and 80% (ES -1.03, CL -1.84, -0.21) 1RM peak power. Differences in VA (ES 0.03, CL -0.77, 0.84) and blood lactate (ES -0.53, CL -1.34, 0.28) were *unclear* between the groups at Pre. The

high volume squatting exercise was effective in causing decreases in markers of fatigue that were *very likely* for MVC (ES -0.96, CL -1.52, -0.39) and VA (ES -1.06, CL -1.63, -0.48), *most likely* for resting doublet force (ES -1.35, CL -1.92, -0.79) and *likely* for 80% 1RM peak power (ES -0.57, CL -1.13, 0.00). Alterations in 20% 1RM peak power were *unclear* compared to Pre (ES -0.24, CL -0.80, 0.33). Blood lactate concentration had *most likely* (ES 2.38, CL 1.82, 2.95) increases after the squatting exercise. After the squatting exercise the middle-aged group showed *very likely* greater decrements in resting doublet force and peak power at 20 and 80% 1RM than the young group (Table 5.3). Between-group differences after the exercise protocol were *unclear* for MVC, VA and blood lactate concentration.

**Table 5.3** Markers of fatigue (mean ± SD) in after squatting exercise in young and middle-aged males. Qualitative descriptor, ES and upper and lower 90% CL are noted in the comparison column.

| Fatigue Indicators | Group | Pre | Post | Comparison |
|---|---|---|---|---|
| MVC (N·m) | *Young* | 265.7 ± 95.8 | 179.2 ± 60.7 | *Unclear* |
| | *Middle-aged* | 199.1 ± 63.3 | 144.9 ± 55.4 | -0.56 (-1.37, 0.25) |
| VA (%) | *Young* | 93.4 ± 5.8 | 85.3 ± 9.4 | *Unclear* |
| | *Middle-aged* | 93.6 ± 5.6 | 82.9 ± 12.9 | -0.20 (-1.00, 0.61) |
| Resting doublet force (N·m) | *Young* | 85.1 ± 10.4 | 64.2 ± 10.4 | *Very likely* ↓ |
| | *Middle-aged* | 69.2 ± 21.1 | 48.3 ± 9.3 | -1.53 (-2.34, -0.71) |
| 20% 1RM peak power (W) | *Young* | 507.9 ± 134.6 | 486.6 ± 112.7 | *Very likely* ↓ |
| | *Middle-aged* | 387.4 ± 87.9 | 357.6 ± 86.2 | -1.21 (-2.03, -0.39) |
| 80% 1RM peak power (W) | *Young* | 1295.3 ± 369.1 | 1098.5 ± 307.1 | *Very likely* ↓ |
| | *Middle-aged* | 977.1 ± 211.1 | 831.9 ± 215.2 | -0.94 (-1.76, -0.12) |
| Blood lactate conc. ($mmol \cdot L^{-1}$) | *Young* | 1.9 ± 0.7 | 9.8 ± 2.9 | *Unclear* |
| | *Middle-aged* | 1.6 ± 0.4 | 8.1 ± 5.2 | -0.39 (-1.18, 0.40) |

*5.3.6 Relationship between internal and external load markers with fatigue*

Only mean HR and OMNI-RPE were related to the muscle function markers for the internal load variables (Table 5.4). That is, mean HR was *likely* (*r* = .45, CL .06, .72) and *very likely* (*r* = .50, CL .13, .75) correlated with decrements in MVC and peak power at 80% 1RM, respectively, while OMNI-RPE was *likely* correlated with alterations in peak power at 20 (*r* = .36, CL -.05, .66) and 80% 1RM (*r* = .32, CL -.09, .64). For external markers of load, changes in mean peak power were *likely* correlated (*r* = .35 to .43) with all decrements in muscle function. Similarly, a higher volume load during the protocol was *very likely* related to changes in the muscle function markers (*r* = .50 to .59).

**Table 5.4** Relationships (qualitative descriptor, ES, upper and lower 90% CL) of internal and external load markers with fatigue.

| | | | **Peak power** | |
|---|---|---|---|---|
| **Load** | **Load markers** | **MVC** | **20% 1RM** | **80% 1RM** |
| | | *Likely* | *Unclear* | *Very likely* |
| | Mean heart rate | .45 (.06, .72) | .28 (-.14, .61) | .50 (.13, .75) |
| | | *Unclear* | *Likely* | *Likely* |
| | Mean OMNI-RPE | -.06 (-.45, .35) | .36 (-.05, .66) | .32 (-.09, .64) |
| | | *Unclear* | *Unclear* | *Unclear* |
| | sRPE | .07 (-.34, .46) | .18 (-.24, .54) | .29 (-.13, .62) |
| | | *Unclear* | *Unclear* | *Unclear* |
| **Internal** | Blood lactate increase | .22 (-.57, 0.2 | -.20 (-.55, .22) | -.19 (-.55, .23) |
| | | *Unclear* | *Unclear* | *Unclear* |
| | Mean peak velocity | -.05 (-.44, .36) | .04 (-.37, .43) | .02 (-.38, .42) |
| | | *Likely* | *Likely* | *Likely* |
| | Mean peak power | .38 (-.03, .68) | .43 (.03, .71) | .35 (-.06, .66) |
| | | *Very likely* | *Very likely* | *Very likely* |
| **External** | Volume load | .59 (.24, .80) | .55 (.19, .78) | .50 (.13, .75) |

## 5.4 Discussion

To the author's knowledge this is the first study to compare internal and external load variables, and fatigue response from squatting exercise, in resistance trained young and middle-aged males. These data indicate that the internal load during squatting exercise at the same relative intensity is not different in these groups, though certain measures of external load (i.e. volume load and peak power) are. Moreover, when compared to younger males, middle-aged males can expect greater decrements in peak power after squatting exercise, which appear to be related to certain internal (HR and OMNI-RPE) and external (peak power and volume load) load measures.

This study recorded *unclear* differences in HR and the HR rate of change during the resistance exercise between the two age groups. These data contrast to previously observed differences in HR between young and older physically active men during isometric knee extension exercise (Smolander et al., 1998), but reaffirm no difference in HR between younger and older males during leg press exercise (Kawano et al., 2008). Similarly, the *unclear* differences observed in OMNI-RPE and the OMNI-RPE rate of change over the resistance exercise protocol are supported by previous data (Manini et al., 2012), but oppose previous findings in young and older males (Allman & Rice, 2003; Justice et al., 2014; Pincivero, 2010). The similar internal responses between groups in the current study might reflect similar alterations in vagal tone and motor command (deMorree, Klein & Marcora, 2012; Smolander et al., 1998) during resistance exercise in young and middle-aged males who regularly resistance train. sRPE was not different between groups after the exercise, which is surprising given that sRPE is related to the volume load (Genner & Weston, 2014) that was moderately higher in the young group. sRPE appears to monitor the participant's perception of the exercise in the context of the physical and psychological state (Impellizzeri et al.,

2004), which indicates that, holistically, the resistance trained young and middle-aged males perceived the exercise similarly. For blood lactate concentrations, differences were *unclear* between groups after resistance exercise. Though higher blood lactate concentrations have been observed in younger compared to older males (Smilios et al., 2007; Walker et al., 2013), the similarities in the current study might suggest a similar reliance on glycolytic pathways during the squatting exercise in the two groups. The current study also observed *unclear* differences in any external to internal load ratios, which would indicate that the internal response for a given external load is similar between young and middle-aged males during squatting exercise. Collectively, these data suggest that internal load markers in young and middle-aged resistance trained males are similar during high volume squatting exercise at the same relative load.

Given that young resistance trained males can produce higher velocities than middle-aged males (Chapter 4) it is perhaps surprising that differences in the peak velocity between groups during the exercise protocol were *unclear*, albeit 60% 1RM for squat demonstrated the lowest differences between groups (ES = 1.0; Chapter 4). Also, the repeated squatting in this chapter, compared to single repetitions performed in Chapter 4, might have been subject to pacing in order to prevent premature fatigue. A further explanation for the differences in velocity during exercise between age groups might come from the participants' familiarity with the movement. For example, Petrella and colleagues (2005) noted greater fatigability and lower velocity in older adults (~64 years) compared to their young (~27 years) counterparts during knee extension exercise, but no differences were present during explosive sit-to-stand exercise. No difference in sit-to-stand exercise was attributed to familiarity with that movement in both groups, i.e. they would perform sit-to-stand movements in their daily

routines whereas the older group were not familiar with knee extension exercise (Petrella et al., 2005). Given that all participants regularly squatted as part of their resistance programmes, this would explain no difference in peak velocity between groups in the current study. Over the exercise protocol, peak power was moderately higher in the young group compared to the middle-aged group while the rate of change in peak power was *unclear* between groups. This supports previous observations of lower power output and similar fatigability during explosive sit-to-stand exercise (Petrella et al., 2005). Interestingly, Petrella and colleagues (2005) noted that differences in power between ages were driven by differences in velocity during exercise, yet the current study observed no differences in velocity. That power is the product of the velocity and force (i.e. the load) would indicate that the differences in peak power in the current study are due to the higher volume load performed by the young males. That is, the differences in power between young and middle-aged resistance trained males during the exercise are a consequence of differences in force (i.e. the volume load) and not velocity as suggested by Petrella et al. (2005) in young and old males. Accordingly, this study indicates that peak power, but not peak velocity, is higher in young compared to middle-aged resistance trained males during high volume squatting exercise.

Reductions in muscle function immediately after the squatting exercise are indicative of fatigue (i.e. inability to maintain the expected force or power output; Edwards, 1981). Lower VA after the squatting exercise suggests that impairments in force and power output were influenced by a reduction in drive to the muscle caused by neural impairments and a reduction in excitability to the alpha motor-neuron (Avela et al., 1999; Horita et al., 1999; Morton et al., 2005). In addition, the lower resting doublet force after exercise indicates peripheral alterations, that is, a disruption of

sarcomeres, impaired E-C coupling and the acculumation of fatigue-related metabolites, for example, inorganic phosphates and hydrogen ions (Allen et al., 2008; Doguet et al., 2016; Hubal et al., 2007) might have also contributed to the reductions in MVC and peak power at 80% 1RM after the squatting. After exercise, resting doublet force and peak power at 20 and 80% 1RM had *very likely* greater decrements in the middle-aged group compared to the young group, where differences in MVC and VA were *unclear*. Greater fatigue in older populations after isoinertial compared to isometric actions are well supported (Avin & Frey Law, 2011; Christie et al., 2011) and may reflect an elevated energy cost of contraction (Layec et al., 2014) and impairments in cross-bridge cycling (Dalton et al., 2012) with age. The greater decrements in resting doublet force in the middle-aged males contrast to the similar reductions between age groups after knee extension exercise reported by Dalton and colleagues (2012) and are indicative of greater peripheral alterations (i.e. disruption of sarcomeres and impaired E-C coupling; Allen et al., 2008; Doguet et al., 2016; Hubal et al., 2007) after high volume exercise. The *unclear* differences between groups in VA are similar to those previously reported by Dalton and colleagues (2012) and suggest comparable central alterations after high volume exercise. As such, middle-aged trained males can expect a similar isometric, but not peak power, fatigue response after high volume squatting exercise.

Mean HR during exercise was moderately correlated with decrements in MVC and 80% 1RM peak power ($r$ = .45 and .50, respectively). It is unknown why a greater cardiovascular load during squatting exercise might result in larger impairments in MVC and peak power at high external loads. Previous work by Rezk and colleagues (2006) noted that elevated HR, albeit after resistance exercise, was associated with a cardiac sympathetic activation and parasympathetic deactivation. Like Rezk et al.

(2006), the higher HR in the current study are likely to driven by alterations in cardiac sympathetic and parasympathetic activity, which aim to increase oxygen delivery to the working musculature. OMNI-RPE was moderately associated with decrements in peak power at both 20 and 80% 1RM (*r* = .36 and .32, respectively). It is suggested that perception of effort reflects central motor command to the muscles (deMorree et al., 2012). Moreover, an increase in central motor command might seek to augment muscle activation in order to lift the load when the muscle is fatiguing (deMorree et al., 2012). Thus, it is understandable that an elevated OMNI-RPE would be associated with reductions in post-exercise fatigue markers. These data indicate a dose-response relationshipbetween HR and OMNI-RPE during high volume resistance exercise and post-exercise decrements in muscle functional markers. Practitioners should be cognisant of the relationship between higher HRs and OMNI-RPEs with post-exercise decrements in muscle function. This study also reported those with a higher volume load were subject to greater impairments in MVC and peak power at 20 and 80% 1RM (*r* = .59, .55 and .50, respectively). These data are similar to previous observations of greater reductions in MVC after lower-limb resistance protocols with a higher amount of work performed (Brandon et al., 2015; Howatson et al., 2016). The moderate correlations with average peak power during exercise and post-exercise reductions in MVC and peak power at 20 and 80% 1RM are the first of their kind. Like the suggestions of Brandon et al. (2015) and Howatson et al. (2016), these reductions in MVC might be owing to metabolic (i.e. increased use of the glycolytic pathway, which is indirectly supported by the higher post-exercise blood lactate) and peripheral alterations (i.e. impaired E-C coupling, demonstrated by the reduction in resting twitch scores after exercise). The relationships between external load (volume load and mean peak power) with post-exercise decrements in peak power during back squat

are novel and indicate that a dose-response relationship exists between these variables. Importantly, these data suggest that the applied practitioner can monitor volume load and mean peak power during resistance exercise should they need to be cognisant of the post-exercise impairments in muscle function after lower-limb exercise.

## 5.5 Conclusion

This study examined the load (internal and external) and fatigue response in young and middle-aged males after high volume squatting exercise. These data indicate that internal load is not different between young and middle-aged resistance trained males during squatting exercise, though certain external load measures (peak power and volume load) are. Practically, these findings suggest that internal, but not external, load can be used to monitor high volume resistance training in a like manner between these age groups. Moreover, high volume squatting exercise impairs peak power at low and high external loads to a greater extent than isometric force in middle-aged males compared to their young counterparts. The applied practitioner should be mindful of these reductions in peak power in middle-aged males and programme lower-body resistance training accordingly. The correlations observed in this study indicate that certain internal (HR and OMNI-RPE) and external (mean peak power and volume load) load are positively related to the post-exercise decrements in muscle function. As such, it is suggested that applied practitioners monitor these variables when post-exercise decrements in muscle function are undesirable.

# 6. Exercise-induced muscle damage and recovery in young and middle-aged males with different resistance training experience

## 6.1 Introduction

Alongside the increasing number of middle-aged (i.e. 30 to 59 year-olds) people in the U.K. (~600,000 projected from 2014 to 2019; Office for National Statistics 2014) is a growing number of middle-aged athletes (Leper et al., 2013; Tanaka & Seals 2008). Many of these athletes want to maintain or improve their athletic performances despite the natural, age-related declines (Baker & Tang 2010; Pantoja et al., 2016). Specifically, these impairments are because of ~1% per annum losses in muscle mass (i.e. sarcopenia; Frontera et al., 2000; Narici et al., 2004; Welle, 2002) and ~1 to 3.5% per annum losses of muscle strength and power (i.e. dynapenia; Candow & Chilibeck, 2007; Clark & Manini, 2012; Frontera et al., 2000; Izqueirdo et al., 1999; Skelton et al., 1994) of which the lower-body undergoes the greatest losses (Candow & Chilibeck, 2005; Chapter 4; Frontera et al., 2000; Noguiera et al., 2013). Importantly, in the applied setting, athletes adopt resistance training strategies, of varying intensities, weekly frequencies and durations, to ameliorate or off-set these age-associated changes (Bottaro et al., 2007; Kongsgaard, et al., 2004; Kosek et al., 2006; Newton et al., 2002; Roth et al., 1999; Sayers & Gibson, 2010; Sayers & Gibson, 2014).

Resistance exercise can result in EIMD (Hortobagyi et al., 1998; Roth et al., 1999), for which the mechanisms have been discussed extensively before (e.g. Damas et al., 2016; Hyldahl & Hubal, 2014; Morgan, 1990; Morgan & Allen, 1999; Proske & Morgan, 2001). The symptoms of EIMD include increases in muscle soreness, intramuscular enzymes in the blood serum and plasma, and of most importance to the athlete, an impaired muscle function that includes reductions in

muscle strength and power (Damas et al., 2016; Flavo & Bloomer, 2006; Hyldahl & Hubal, 2014) that can negatively affect sporting performance. These symptoms typically peak between 24 and 48 hours after the initial bout and are recovered by seven days (Damas et al., 2016; Flavo & Bloomer, 2006; Hyldahl & Hubal, 2014). The appearance of these symptoms is not synchronous and often highly individualised (Clarkson et al., 2005; Hubal, Rubenstein & Clarkson, 2007; Machado & Willardson, 2010). A muscle's susceptibility to damage might also be affected (reduced) in subsequent bouts where prior eccentric exercise has occurred (Hyldahl, Chen & Nosaka, 2017; Nosaka, et al., 2001). Several studies have noted that this protection from eccentric exercise is less pronounced (~29% in MVC) in older compared to younger men (Lavender & Nosaka, 2006b; Gorianovas et al., 2013), which suggests that older resistance trained men might exhibit symptoms of EIMD that are not dissimilar to their untrained counterparts.

Studies examining recovery of older and younger untrained adults after muscle-damaging exercise are equivocal. Some have reported greater symptoms of EIMD in younger (~20 years) compared to older males (~71 years; Lavender & Nosaka, 2006a; Lavender & Nosaka, 2007), while others have observed greater EIMD in older (~59 to 66 years) compared to younger males (~23 years; Manfredi et al., 1990) and females (20 to 30 years; Ploutz-Snyder et al., 2001). These are also in contrast to studies reporting no difference in symptoms of EIMD after exercise for young (~19 years) compared to middle-aged (~48 years; Gordon et al., 2017; Lavender & Nosaka, 2008) or older males (~64 to 76 years Buford et al., 2014; Chapman et al., 2008; Clarkson & Dedrick, 1988; Roth et al., 1999). If these symptoms, particularly the loss of strength, are different between age groups, it would be beneficial to gain an insight into the mechanisms of these changes. Buford et al. (2014) were the first to investigate the

acute effects of eccentric unilateral plantar flexion exercise among young (~23 years) and older (~76 years) adults and reported that recovery from EIMD was not different between the two groups when controlling for general physical activity levels. More recently, Gordon and colleagues (2017) observed no differences in indirect markers of EIMD between recreationally trained young (~22 years) and middle-aged (~47 years) males after damaging knee extensor exercise. Despite these novel findings, no study has yet reported on the recovery characteristics from multi-jointed lower-body exercise in middle-aged (35 to 55 years), resistance trained males. Indeed, Gordon et al. (2017) advised that future studies might adopt a more ecologically valid exercise protocol. Data from such a study would be highly applicable to those athletes seeking to prolong their careers. Consequently, the primary aim of the study was to determine the time course to recovery from EIMD in young and middle-aged resistance trained males. A secondary purpose was to determine if the recovery profile of middle-aged males is altered by resistance training experience.

## 6.2 Methods

### *6.2.1 Participants*

Nine young resistance trained (YG; range: 21 to 25 years), nine middle-aged (MT; range: 35 to 54 years) resistance trained, and nine untrained middle-age males (MU; range: 35 to 53 years) were recruited for this study using convenience sampling. Participants were from the University population, local gymnasia and sports teams. Thirty-five years was selected as the lower boundary for the middle-aged group because it is the entry age for ‘Masters’ athletes (see British Masters Athletic Federation and World Masters Athletics). As age-related studies typically use older groups (60 years and over), 55 was selected as the upper-limit for the middle-aged

group. An overall sample size of approximately 27 (nine per group) was estimated using Batterham and Atkinson's (2005) nomogram. This was calculated using a CV and typical change of 6.1% (the inter-day reliability for 80% 1RM) squat observed in Chapter 3) and 5%, respectively. The YG and MT had a minimum of two years' resistance training experience and regularly used squats as part of their resistance training programmes. The MU group had no resistance training experience but was screened by the lead researcher to ensure they could perform the correct squat technique. All participants had been active in sport for a minimum of two years. Participants completed a pre-test health questionnaire and provided written consent for the study, which was approved by the Ethics Committee of the Faculty of Life Sciences at the host institution. Participants were instructed not to consume any ergogenic supplements (for example, caffeine) on the day of testing and to refrain from exercise, other than that performed as part of the study, throughout their involvement.

### *6.2.2 Study design*

The study used a mixed factorial design (age group x trials) whereby participants attended the laboratory on four separate occasions, the initial visit for estimations of body composition and back squat 1RM. On the same visit they were habituated with the measurements of squatting peak power and MVC, VA and resting doublet force during isometric knee extension. Participants were considered 'habituated' when they could complete three consecutive repetitions that produced peak powers or torque values each within 10% (Batterham & George, 2003). Participants returned to the laboratory 2-4 days later for pre-measurements (peak power during squat at 20 and 80% 1RM, MVC and VA, muscle soreness and CK activity) and an exercise bout comprising 10 x 10 squats at 60% 1RM (MacDonald et al. 2014). Repeated measures

(peak power testing during squat at 20 and 80% 1RM, MVC, VA and resting doublet force, muscle soreness and CK) were conducted 24 and 72 hours after the initial exercise bout.

### *6.2.3 Procedures*

#### *6.2.3.1 Anthropometric measurements*

Body density was estimated via skinfold thickness measurements (Harpenden, British Indicators, Burgess Hill, UK) taken at the tricep, axilla, abdominal, suprailiac, chest, subscapular, and mid-thigh incorporated into the equation of Jackson and Pollock (1978). Body fat percentage was derived from the equation (Heyward & Wagner, 2004):

$$\text{Body fat percentage} = [(5.21/ \text{body density}) - 4.78] \times 100$$

From this, quantities (kg) of FM and FFM were derived for each group.

#### *6.2.3.2 Resistance training history and sports participation*

The YG and MT participants completed a questionnaire to record how many years they had participated in regular resistance training, their weekly training frequency and session duration, and the main reason for their training. A second questionnaire detailed how many years they had participated in organised sport, their weekly frequency and session duration and the type of sport they participated in (i.e. team, endurance, racket or other).

#### *6.2.3.3 Maximal strength testing*

To avoid the risk associated with maximal strength testing (Mazur et al. 1993), 1RM for squat exercise was predicted using a 3RM protocol. In brief, participants performed

8-10 repetitions with 50% of their estimated 1RM, followed by 3-5 repetitions with 85% of their estimated 1RM. The load was then set at the approximate 1RM and the participants performed one repetition. The load was progressively increased until the participant could no longer perform a complete repetition. The final load lifted was then used with the following equation (Wathan, 1994) to estimate 1RM squat load:

$$1RM = (100 \times 3RM \text{ load lifted})/[48.8 + (53.8 \times 2.71828^{-0.075} \times \text{repetitions})]$$

The above equation has been reported to yield accurate 1RM predictions ($r$ = 0.969, 0.02% different from direct 1RM; LeSuer et al., 1997).

*6.2.3.4 Assessment of perceived muscle soreness*

Perceived muscle soreness of the knee extensors was measured using a visual analogue scale (VAS). The VAS is numbered from 0 to 10 (on the concealed reverse side of the scale) where 0 indicates “no soreness on movement” and 10 indicates “muscles are too sore to move”. With feet shoulder width apart and hands on hips, participants descended until their hips were below their knee joint at which point the VAS was held up for them to indicate their rating of perceived soreness on the continuum. This method has been used previously as an indirect marker of muscle damage (e.g. Burt & Twist, 2011; Burt et al., 2014).

*6.2.3.5 Assessment of plasma creatine kinase activity*

Plasma CK activity was determined from a capillary blood sample of the participant’s preferred finger. A 30 µl sample of whole blood was collected into a heparinised capillary tube and pipetted onto a test strip for analysis (Reflotron, Type 4, Boehringer

Mannheim, Mannheim, Germany). The Reflotron employs a photometric process to determine CK activity. This procedure has been used in several previous studies to confirm tissue damage after exercise (e.g. Burt et al., 2014; Davies, Rowlands, Poole, Jones & Eston, 2011).

*6.2.3.6 Assessment of peak power during squat*

Peak muscle power was assessed at loads corresponding to 20 and 80% 1RM during back squat exercise using a rotary encoder (FitroDyne, Fitronic, Bratislava, Slovakia) attached via a nylon cord directly under a Smith machine bar (Perform Better, Leicester, UK). As the FitroDyne measures rate of displacement and assumes that the nylon cord is moving in a vertical plane, a Smith machine was used to prevent deviation from this plane and decrease measurement error. The FitroDyne has been shown to produce reliable intra- and inter-day measures of peak power (CVs = 3.9-6.1%) at the selected loads (Chapter 3).

With the bar positioned across the shoulders, participants squatted until their hips were below the knee joint and then ascended as rapidly as possible until their knees were at full extension. A bench was employed to ensure that they attained the same depth and range of motion on each repetition. Three repetitions at each load were performed with self-selected rest intervals that ranged from 30 to 90 s (Chapter 3). Rest times were self-selected, as lighter loads (20% 1RM) did not require the same recovery time. Peak velocity was recorded from which peak power was calculated (load x velocity x 980 $cm \cdot s^{-1}$)/100). The load order was randomised for each participant to negate possible ordering effects.

*6.2.3.7 Assessment of maximal voluntary contraction and voluntary activation*

Before undertaking the MVC and VA assessments, participants performed a warm-up comprising five minutes of cycling at 100 W (Lode, Corival, Groningen, Netherlands). A dynamometer (Biodex, Multi-joint system 3, Biodex Medical, New York, USA) was used to measure isometric force of the participant's dominant knee extensors at 80° knee flexion. To prevent extraneous body movements, Velcro straps were applied tightly across the chest and thigh. Participants were provided with strong verbal encouragement and real-time feedback via the PC monitor.

The knee extensors were electrically stimulated (5 s with two 100 Hz single square impulses (doublet); Digitimer, D57, Hertfordshire, UK) using two 5 x 13 cm moistened surface electrodes (Axelgaard Manufacturing Co LTD, Fallbrook, CA); one placed distally over the quadriceps and the other proximally over the upper quadriceps. During optimisation the amplitude of a doublet was progressively increased, starting at 50 amps, until a point where no further increases in intensity resulted in an increase in resting doublet force. Initially a 230 volt electrically evoked doublet (set 20% above the value required to evoke a resting muscle doublet of maximum amplitude) was applied to the resting muscle (resting doublet) at 1 s. The resting doublet was used to elucidate any peripheral alterations that might have occurred as a result of the squatting protocol (MacDonald et al., 2014). Participants then performed a 4 s MVC before a doublet which was applied at the isometric plateau (superimposed doublet). The MVC was taken as the average force over 50 ms (AcqKnowledge 3 software, Biopac Systems, Massachusetts) before the superimposed doublet was applied. VA was calculated according to the interpolated twitch ratio (Merton, 1954) using the equation;

VA (%) = [1- (size of superimposed doublet/ size of resting doublet)] x 100

A similar procedure has been deemed a reliable method (CV = 3.38%) for assessing VA (Morton et al., 2005).

*6.2.3.8 Muscle-damaging exercise protocol*

This consisted of 10 sets of 10 repetitions of squat exercise at a load corresponding to 60% 1RM with 120 s rest between sets (MacDonald et al., 2014). For each repetition participants descended for 3 s until their hips were below the knee joint and then ascended as rapidly as possible until their knees reached full extension. A bench was employed to standardise the depth of each repetition. A similar protocol has successfully induced symptoms of muscle damage in previous research (Burt et al., 2012; Burt et al., 2014; MacDonald et al., 2014). The FitroDyne was used to calculate power for each repetition in the manner outlined previously. Average peak power per repetition was used to elucidate the influence of exercise intensity on recovery profiles between groups. One participant from the MU group was unable to complete sets 8, 9 and 10 at 60% 1RM, thus the load was reduced by 5 kg (50.1% 1RM) and power values were calculated accordingly.

*6.2.4 Statistical analysis*

Comparisons of categorical training history and sport participation variables (i.e. weekly training and sports frequency, session duration, reason for training and type of sport) by group were made using a chi-squared ($\chi^2$) test of association. All other data were analysed using the ES with 90% CL (Hopkins, et al. 2009). Magnitude-based inference statistics were used to provide information on the size of the differences,

allowing for a more practical and meaningful explanation of the data (Batterham & Hopkins 2006). Thresholds for the magnitude of the observed change for each variable were determined as the within-participant standard deviation in that variable x 0.2, 0.6, 1.2 and 2 for a small, moderate, large and very large effect, respectively (Cohen, 1988). Threshold probabilities for a meaningful effect based on the 90% CL were: <0.5% *most unlikely*, 0.5–5% *very unlikely*, 5–25% *unlikely*, 25–75% *possibly*, 75–95% *likely*, 95–99.5% *very likely*, >99.5% *most likely*. Effects with CL across a likely small positive or negative change were classified as *unclear* (Hopkins et al., 2009). All calculations were completed using predesigned spreadsheets (Hopkins, 2006; Hopkins, 2017). Data are presented as ES, lower CL and upper CL.

## 6.3 Results

### *6.3.1 Biometric measures and training history*

Age and sum of skinfolds were *most likely* and *likely* higher, respectively, in the MT groups compared to the YG group (Table 6.1). Differences in FM and body fat percentage between the YG and MT groups were *very likely*, while mass and squat 1RM were *unclear*. Age and FFM differences between the MT and MU groups were *likely moderate*, whilst all other biometric characteristics demonstrated *unclear* differences.

The MT group had *most likely* regularly resistance trained for longer than the YG (ES 2.29, CL 1.46, 3.13; Table 6.2), though their training was associated with a lower weekly frequency ($\chi^2$ = 32.5, $P$ < 0.05) and shorter session duration ($\chi^2$ = 36.4, $P$ < 0.05). Moreover, the MT group typically chose resistance training for strength and fat loss, whereas the YG trained for strength ($\chi^2$ = 31.8, $P$ < 0.05).

**Table 6.1** Biometric characteristics (mean ± SD) and comparisons of young (YG) and middle-aged trained (MT) and untrained (MU) groups. Qualitative descriptor, ES and upper and lower 90% CL are noted in the comparison column.

| | Group | | | Comparison | |
|---|---|---|---|---|---|
| Measure | YG (*n* = 9) | MT (*n* = 9) | MU (*n* = 9) | YG v MT | MT v MU |
| Age (y) | 22.3 ± 1.7 | 39.9 ± 6.2 | 44.4 ± 6.3 | *Most likely* ↑<br>3.70 (2.87, 4.53) | *Likely* ↑<br>0.71 (-0.10, 1.52) |
| Mass (kg) | 82.0 ± 9.0 | 79.1 ± 10.3 | 83.4 ± 9.56 | *Unclear*<br>0.29 (-1.10, 0.52) | *Unclear*<br>0.42 (-0.39, 1.23) |
| Fat-free mass (kg) | 71.4 ± 7.9 | 63.9 ± 6.5 | 68.6 ± 7.1 | *Very likely* ↓<br>-1.02 (-1.83, -0.22) | *Likely* ↑<br>0.68 (-0.13, 1.49) |
| Fat-mass (kg) | 10.5 ± 4.5 | 15.2 ± 5.7 | 14.8 ± 7.0 | *Likely* ↑<br>0.89 (0.09, 1.70) | *Unclear*<br>-0.07 (-0.88, 0.74) |
| Body fat (%) | 12.8 ± 4.7 | 18.8 ± 5.8 | 17.4 ± 6.7 | *Very likely* ↑<br>1.13 (0.32, 1.94) | *Unclear*<br>-0.23 (-1.04, 0.58) |
| Sum of skinfolds (mm) | 82.3 ± 24.6 | 102.4 ± 31.9 | 91.7 ± 32.7 | *Likely* ↑<br>0.69 (-0.12, 1.50) | *Unclear*<br>-0.32 (-1.13, 0.48) |
| Squat 1RM (kg) | 130.8 ± 26.8 | 109.3 ± 22.5 | 98.4 ± 14.25 | *Likely* ↓<br>-0.85 (-1.65, -0.04) | *Unclear*<br>-0.56 (-1.37, 0.25) |

**Table 6.2** Resistance training characteristics of the young (YG) and middle-aged trained groups (MT).

| | YG (*n* = 9) | MT (*n* = 9) |
|---|---|---|
| **Years of resistance training** | | |
| (mean ± SD) | 4.6 ± 1.3 | 18.0 ± 5.6 |
| **Weekly frequency*** | | |
| *1 to 2* | 2 (22.2) | 6 (66.7) |
| *3 to 4* | 4 (44.4) | 2 (22.2) |
| *5+* | 3 (33.3) | 1 (11.1) |
| **Session duration*** | | |
| *0 to 30 minutes* | 0 (0.0) | 1 (11.1) |
| *31 to 60 minutes* | 3 (33.3) | 7 (77.8) |
| *61 to 90 minutes* | 5 (55.6) | 1 (11.1) |
| *90+ minutes* | 1 (11.1) | 0 (0.0) |
| **Reason for resistance training*** | | |
| *Strength* | 6 (66.7) | 4 (44.4) |
| *Hypertrophy* | 1 (11.1) | 0 (0.0) |
| *Fat loss* | 1 (11.1) | 4 (44.4) |
| *Health* | 1 (11.1) | 1 (11.1) |

*categorical variables are significantly associated ($P < 0.05$). Brackets denote percentage of responses in each category.

There were *very likely large* and *moderate* differences in sports participation for the MT compared to the YG and MU, respectively, with MT having more years compared to the YG (ES 1.47, CL 0.66, 2.28) and less than the MU group (ES 1.17, CL 0.36, 1.98; Table 6.3). No relationship ($P > 0.05$) was observed between groups for weekly frequency, session duration or type of sport played.

**Table 6.3** Sports participation characteristics of the young and middle-aged trained groups.

| | YG (*n* = 9) | MT (*n* = 9) | MU (*n* = 9) |
|---|---|---|---|
| **Years of sports participation** (mean ± SD) | 11.2 ± 4.8 | 22.0 ± 7.8 | 30.3 ± 7.8 |
| **Weekly frequency** | | | |
| *1 to 2* | 4 (44.4) | 2 (22.2) | 0 (0.0) |
| *3 to 4* | 4 (44.4) | 4 (44.4) | 6 (66.7) |
| *5+* | 1 (11.1) | 3 (33.3) | 3 (33.3) |
| **Session duration** | | | |
| *0 to 30 minutes* | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| *31 to 60 minutes* | 3 (33.3) | 4 (44.4) | 7 (77.8) |
| *61 to 90 minutes* | 3 (33.3) | 3 (33.3) | 1 (11.1) |
| *90+ minutes* | 3 (33.3) | 2 (22.2) | 1 (11.1) |
| **Type of sport** | | | |
| *Team* | 5 (55.6) | 3 (33.3) | 3 (33.3) |
| *Endurance* | 3 (33.3) | 5 (55.6) | 4 (44.4) |
| *Racket* | 0 (0.0) | 1 (11.1) | 2 (22.2) |
| *Other* | 1 (11.1) | 0 (0.0) | 0 (0.0) |

*6.3.2 External load response during the muscle-damaging protocol*

There was a *likely moderate* lower average peak power (ES -0.71 CL -1.53, 0.10) in the MT (603.2 ± 162.6 W) compared to the YG (770.4 ± 278.0 W). Differences between the MT and MU (547.0 ± 75.0 W) groups were *unclear* (ES -0.43, CL -1.25, 0.39).

*6.3.3 Perceived muscle soreness*

At Pre, differences between the YG and MT and MT and MU were *unclear* (ES 0.00, CL -0.81, 0.81 and ES 0.42, CL -0.39, 1.22, respectively; Figure 6.1). When the three groups were combined, perceived muscle soreness demonstrated *most likely very large* (ES 4.20, CL 3.74, 4.65) increases at 24 h and likewise (ES 1.82, CL 1.36, 2.27) at 72 hours after muscle-damaging exercise. Between-group differences for the YG

and MT comparison were *unclear* at 24 and 72 h after muscle-damaging exercise. Increases in perceived muscle soreness were *likely moderately* higher in the MU group compared to the MT group at 24 and 72h.

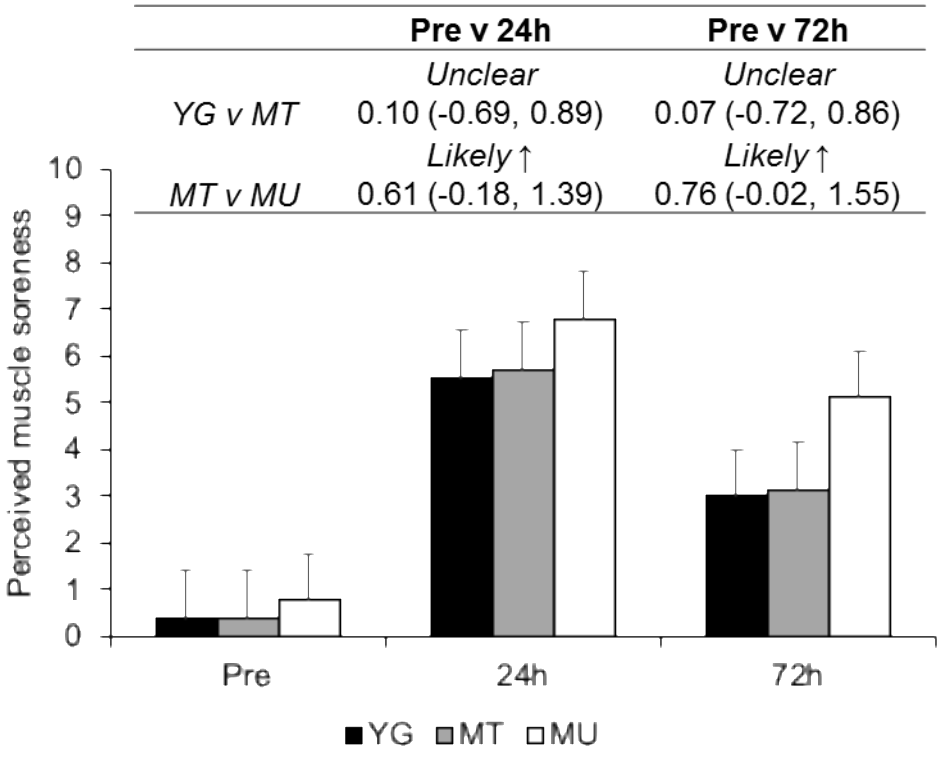


**Figure 6.1** Changes in perceived muscle soreness between YG, MT and MU at pre, 24 and 72 hours after resistance exercise. The panel above details the qualitative descriptor, ES and upper and lower CL.

*6.3.4 Plasma creatine kinase activity*

Differences at Pre for YG and MT and MT and MU comparisons were *unclear* (ES -0.41, CL -1.21, 0.40 and ES -0.44, CL -1.25, 0.38, respectively; Figure 6.2). The increase in plasma CK activity for the three groups combined was *very likely moderate* (ES 1.19, CL 0.73, 1.64) and *likely small* (ES 0.59, CL 0.13, 1.05) at 24 and 72 h, respectively, compared to Pre. Differences in plasma CK activity over time were

*unclear* between the YG and MT groups. Plasma CK activity was *likely moderately* higher in the MU group compared to the MT group at 24 h, though differences between groups were *unclear* at 72 h.

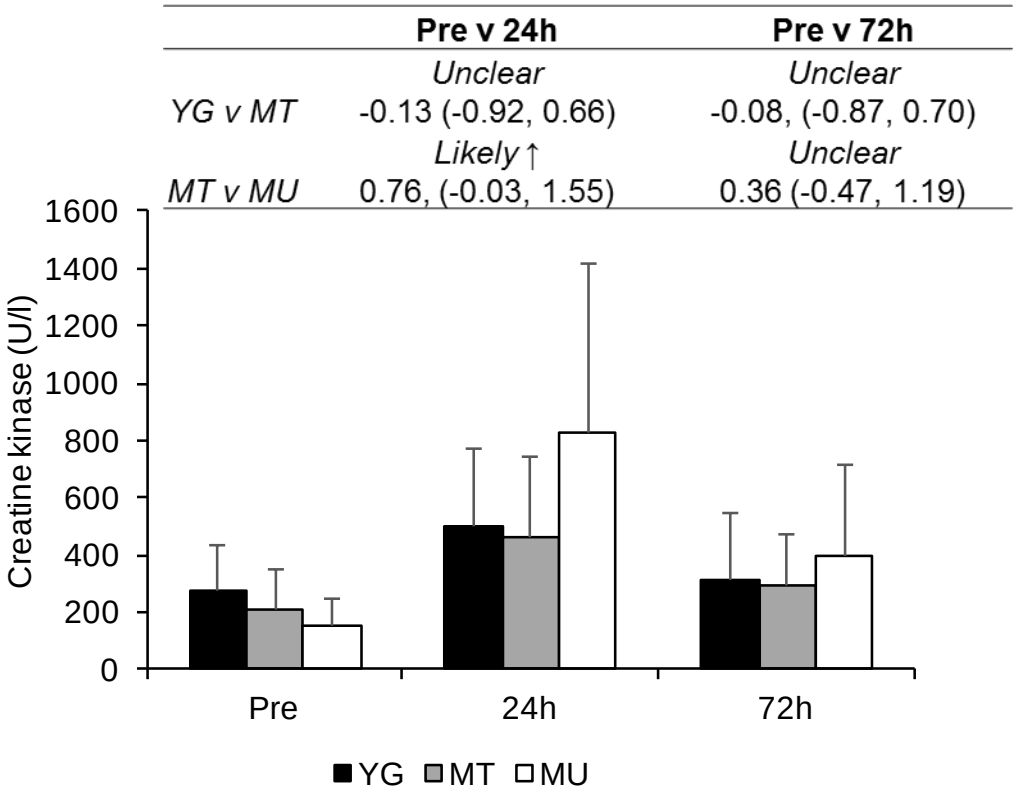


**Figure 6.2** Changes in plasma CK activity between YG, MT and MU at Pre, 24 and 72 hours after resistance exercise. The panel above details the qualitative descriptor, ES and upper and lower CL.

*6.3.5 Peak power during squat exercise*

At Pre, a *very likely moderate* lower peak power was at 20 and 80% 1RM (ES -1.03, CL -1.84, -0.22 and ES -1.03, CL -1.84, -0.21, respectively) was observed in the MT compared to YG (Table 6.4). Differences at Pre were *most likely* higher and *unclear* in the MT group than MU for 20 and 80% 1RM (ES -3.34, CL -4.18, -2.50 and ES -0.47, CL -1.28, 0.33, respectively). When all groups were combined, peak power for

20 and 80% 1RM demonstrated *possibly small* (ES -0.25, CL -0.71, 0.20 and ES -0.36, CL -0.81, 0.09, respectively) and *unclear* (ES -0.23, CL -0.69, 0.22 and ES -0.19, CL -0.64, 0.26, respectively) decrements at 24 and 72 h, respectively. For 20 and 80% 1RM, between-group differences at 24 and 72 h were *very likely* greater in the MT than YG group. Similarly, reductions in 20% 1RM peak power at 24 and 72 h for the MT v MU comparison were *very likely* greater in the MU group. Peak power at 80% 1RM illustrated *likely* and *very likely* greater decrements in the MU than MT group at 24 and 72 h, respectively.

**Table 6.4** Peak power at Pre, 24 and 72 hours. Qualitative descriptor, ES and upper and lower 90% CL are noted in the comparison column.

| | | | | | **Comparison (90% CL)** | |
|---|---|---|---|---|---|---|
| Intensity | Group | Pre | 24h | 72h | Pre v 24h | Pre v 72h |
| **20% 1RM (W)** | *YG* | 507.9 ± 134.6 | 473.8 ± 119.9 | 476.6 ± 119.7 | *YG v MT* | |
| | | | | | *Very likely ↓* | *Very likely ↓* |
| | *MT* | 387.4 ± 87.9 | 360.3 ± 76.1 | 366.3 ± 76.4 | *-1.07( -1.85, -0.28)* | *-1.04 (-1.82, -0.25)* |
| | | | | | *MT v MU* | |
| | *MU* | 320.7 ± 47.9 | 291.7 ± 40.1 | 289.7 ± 40.2 | *Very likely ↓* | *Very likely ↓* |
| | | | | | *-1.06 (-1.84, -0.27)* | *-1.17 (-1.96, -0.39)* |
| **80% 1RM (W)** | *YG* | 1295.3 ± 369.1 | 1207.5 ± 328.2 | 1275.9 ± 338.3 | *YG v MT* | |
| | | | | | *Very likely ↓* | *Very likely ↓* |
| | *MT* | 977.1 ± 211.1 | 869.8 ± 195.0 | 964.9 ± 212.1 | *-1.07 (-1.96, -0.39)* | *-1.04 (-1.83, -0.25)* |
| | | | | | *MT v MU* | |
| | *MU* | 886.0 ± 163.2 | 746.7 ± 153.3 | 735.1 ± 134.8 | *Likely ↓* | *Very likely ↓* |
| | | | | | *-0.67 (-1.45, 0.12)* | *-1.22 (-2.01, -0.43)* |

*6.3.6 Maximal voluntary contraction torque*

At Pre, differences in MVC torque were *likely moderate* and *unclear* for the YG compared to MT (ES -0.80, CL -1.61, 0.01) and MT compared to MU (ES 0.27, CL -0.56, 1.10), respectively (Figure 6.3). MVC torque had *very likely moderate* (ES -0.71, CL -1.16, -0.26) and *likely small* (ES -0.39, CL -0.84, 0.06) decreases at 24 and 72 h after muscle-damaging exercise. *Likely* and *very likely moderate* reductions in MVC torque were observed in the MT group compared to the YG groups at 24 and 72 h, respectively. At 24 and 72 h, differences between the MT and MU groups were *unclear.*

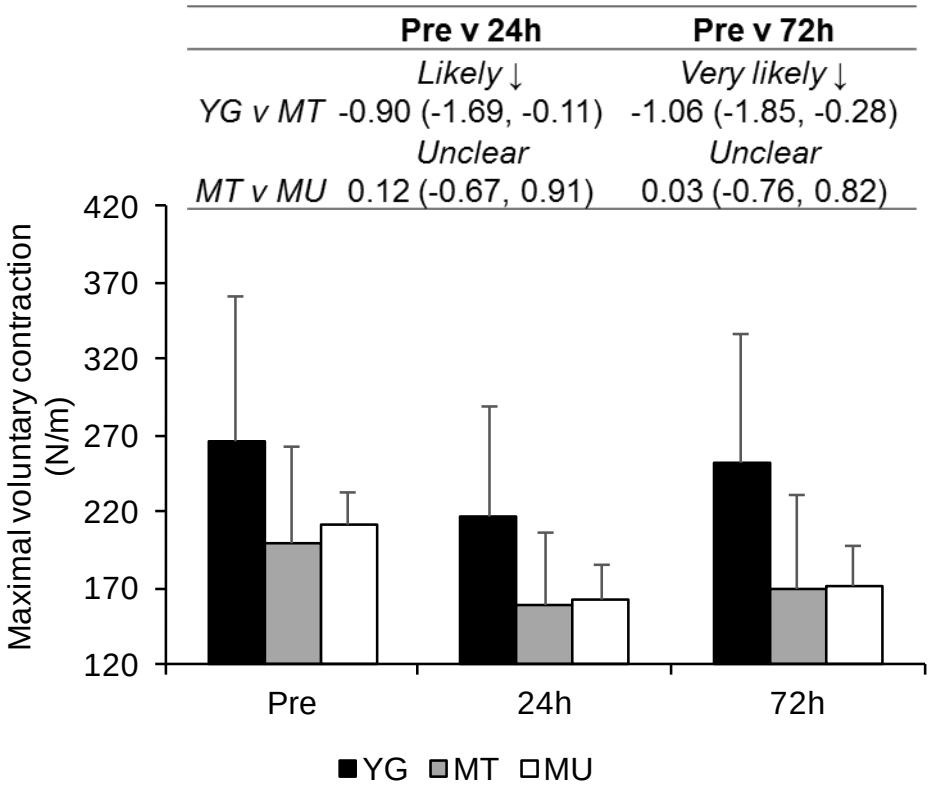


**Figure 6.3** Changes in maximal voluntary contraction torque between YG, MT and MU at Pre, 0, 24 and 72 hours after resistance exercise. The panel above details the qualitative descriptor, ES and upper and lower CL.

*6.3.7 Voluntary activation*

Differences in VA at Pre were *unclear* for YG compared to MT (ES 0.03, CL -0.77, 0.84) and MT compared to MU (ES 0.07, CL -0.76, 0.90; Figure 6.4). When all groups were combined VA decreased over time, with values at 24 and 72 h demonstrating *very likely moderate* decreases (ES -0.87, CL -1.33, -0.41 and ES -0.88, CL -1.34, -0.41, respectively). Differences between groups were *unclear* at all time-points.

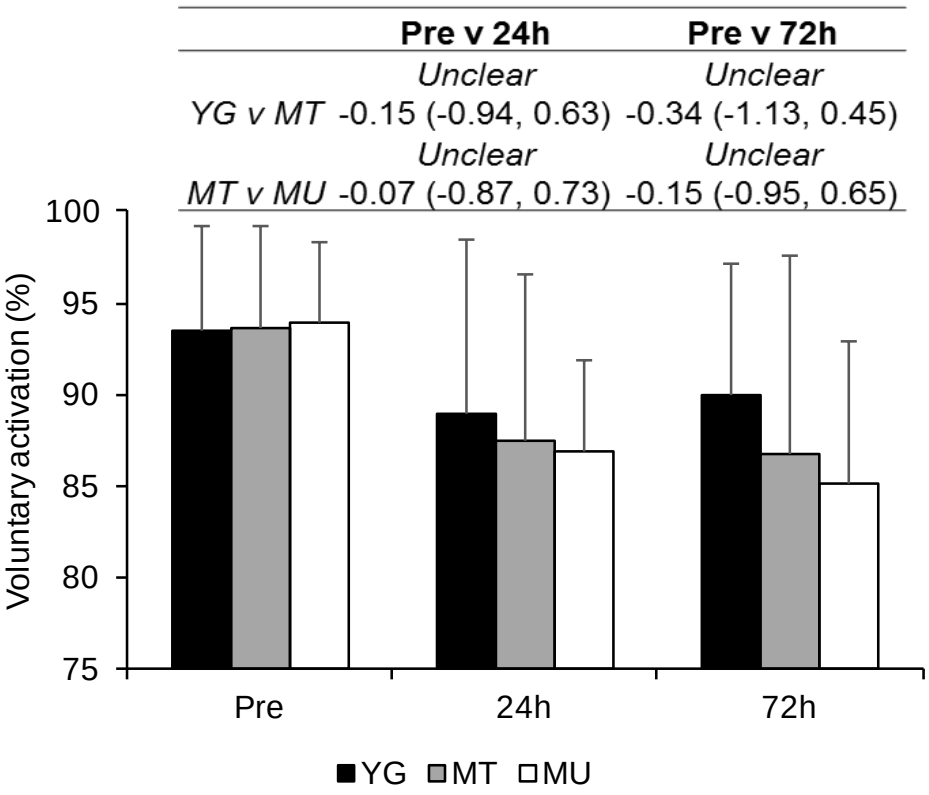


**Figure 6.4** Changes in voluntary activation between YG, MT and MU at Pre, 24 and 72 hours after resistance exercise. The panel above details the qualitative descriptor, ES and upper and lower CL.

*6.3.8 Resting doublet force*

Higher mean resting doublet force values for the YG were *likely moderate* compared to the MT (ES -0.96 CL -1.77, 0.14; Figure 6.5). Similarly, higher values for MU (ES

0.95, CL 0.12, 1.78) where *likely moderate* compared to the MT group. Mean doublet values were *likely small* and *unclear* at 24 and 72 h, respectively, (ES -0.52, CL -0.98, -0.06 and ES -0.04, CL -0.50, 0.42, respectively) after squatting exercise. Differences in resting doublet force were *very likely* and *likely* greater in the MT than YG groups at 24 and 72 h, respectively. MT and MU comparisons were *unclear* at 24 and 72 h.

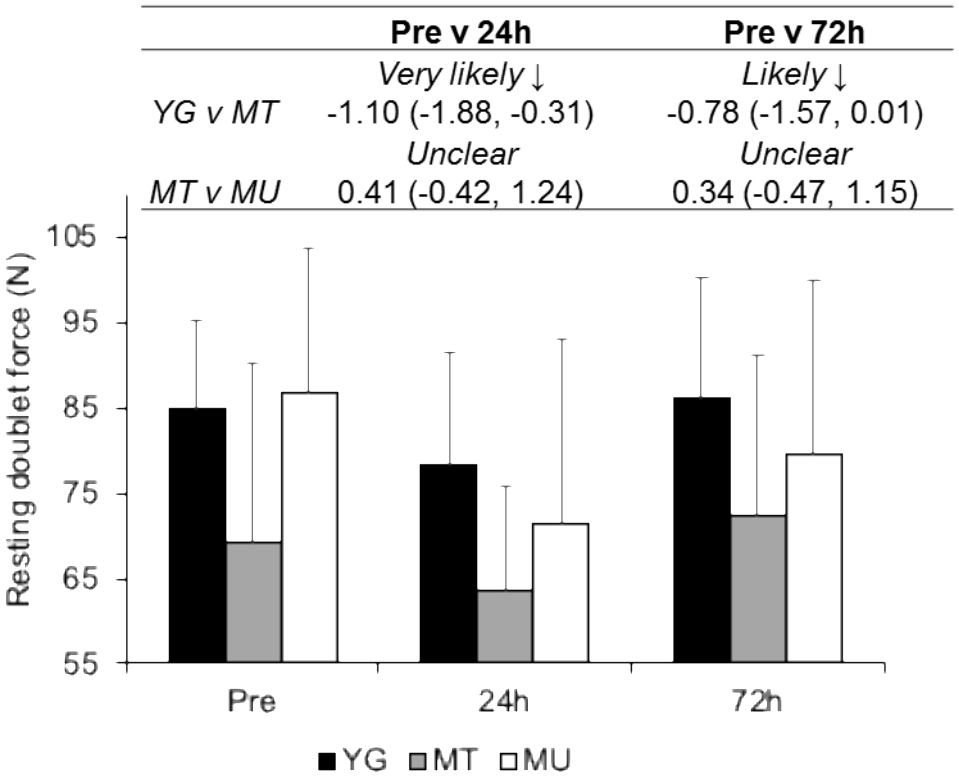


**Figure 6.5** Changes in resting doublet force between YG, MT and MU at Pre, 24 and 72 hours after resistance exercise. The panel above details the qualitative descriptor, ES and upper and lower CL.

### 6.4 Discussion

In the first study of its kind, the current findings highlight the magnitude of EIMD and time-course of recovery after lower-body resistance exercise is greater in trained middle-aged males than their young counterparts. Moreover, regardless of resistance

training experience, middle-aged males experienced like symptoms of muscle damage and a similar recovery profile in the days after.

The small to very large increases in muscle soreness and CK activity and the small to moderate loss of force at 24 and 72 h observed in the current study confirm that the prescribed lower-body resistance exercise caused EIMD. The losses in MVC support previous observations of isometric strength loss after lower-body eccentric exercise (Byrne & Eston, 2002; MacDonald et al., 2014). The reductions in MVC at 24 hours are possibly owing to both peripheral and central impairments, given the contemporaneous decrements in resting twitch and VA. However, that resting doublet scores were recovered by 72 hours but VA remained suppressed suggests that the reductions in MVC at the later time point were caused by central alterations. Potential central mechanisms include a reduction in drive to the muscle caused by neural impairments and reduction in excitability to the alpha motor-neuron (Avela et al., 1999; Horita et al., 1999; Morton et al., 2005).

That differences between groups on plasma CK activity after resistance exercise were unclear reaffirms the findings of previous studies (Buford et al., 2014; Lavender & Nosaka, 2007; Lavender & Nosaka, 2008; Manfredi et al., 1991), suggesting that membrane permeability is similar between young and middle-aged groups. Likewise, the comparable changes in muscle soreness observed in the two resistance trained groups is consistent with the work of Buford et al. (2014) in the plantar flexors, though contradictory to reports of greater soreness experienced by younger males after muscle-damaging elbow flexor exercise (Chapman et al., 2008; Lavender & Nosaka, 2006). Increases in muscle soreness might reflect damage to connective tissue and decreases in range of motion rather than damage to the contractile machinery *per se* (Damas et al., 2016; Hyldahl & Hubal, 2014).

Consequently, these data indicate that CK and muscle soreness responses to lower-limb muscle damaging exercise are similar in young and middle-aged resistance trained males.

Reductions in MVC, VA and resting doublet occurred in both resistance trained groups after EIMD. The finding that Pre VA values were not different between groups contrasts previous suggestions that older athletes are unable to activate the muscle to the same extent as their young counterparts (Klass, Baudry & Duchateau, 2007), possibly owing to the trained nature of the MT group (Knight & Kamen, 2001). That the time course of VA recovery after high volume squatting exercise was no different in the MT and YG groups is also a novel finding. The moderately greater reductions in MVC in the MT group compared to the YG group after EIMD appear to be mediated by peripheral alterations (i.e. disruptions of sarcomeres and impaired E-C coupling), as reflected by the lower resting doublet values in the older trained participants. Given that differences in VA were unclear between the resistance trained groups after EIMD suggests that central alterations are not responsible for the greater reductions in MVC in the MT group.

The lower Pre peak power values at 20 and 80% 1RM in the MT group compared to the YG group are similar to those previously reported (Chapter 4). For the first time, this study has highlighted that the decrements in peak power after EIMD are of a greater magnitude in middle-aged compared to young resistance trained males. Given that lower-body power has strong relationships with a variety of sporting tasks (Cronin & Hansen, 2005; Delaney et al., 2015; Lopez-Segovia et al., 2014; Loturco et al., 2016), it is plausible that the impaired peak power due to EIMD may inhibit these movements. Applied practitioners should therefore be cognisant of this and consider adopting different recovery practices for young and middle-aged male

athletes after muscle-damaging lower-limb exercise. That is, middle-aged males should allow for a greater time for recovery or use recovery aids (e.g. protein strategies).

Both the middle-aged untrained and resistance trained men produced similar peak power during the muscle-damaging protocol that was followed by unclear differences in MVC, VA and resting doublet and likely and unclear differences in CK at 24 and 72 h, respectively. The resistance trained males should experience less muscle damage after eccentric exercise compared to untrained males (McHugh, 2003; Hyldahl et al., 2017; Hyldahl & Hubal, 2015), albeit this protective effect is attenuated in older groups (Lavender & Nosaka, 2006a; Gorianovas et al., 2013). This might explain the similar recovery profiles of CK, MVC, VA and resting doublet in the middle-aged groups reported here. The similar training volume and type of sports played by the two groups might also explain why both groups differ. That is, the training experienced by both groups during their sports participation might have provided a similar protection to the muscle-damaging squatting exercise. A further explanation might be owing to the unclear differences in peak power during the muscle-damaging protocol. It has been noted previously that the magntidue of EIMD and recovery were positively correlated to the workload during the muscle damaging protocol (Toft et al., 2002). Given that both groups produced a similar peak power during the exercise protocol it is not unexpected that the recovery profile was similar. After muscle damaging exercise differences between middle-aged groups in perceived muscle soreness and peak power were moderate to large. The lower muscle soreness in the MT compared to the MU could be due to their experience of muscle soreness during their resistance training i.e. they are accustomed to the feelings of soreness and therefore report lower values. Likewise, the resistance training of the MT group might

preserve or enhance cross-sectional area of the type 2 fibres (Verdijk et al., 2009), thus accounting for their smaller losses in peak power after muscle-damaging exercise. Consequently, resistance training in middle-aged males might help to maintain lower-body peak power after muscle-damaging exercise but does not appear to alter other indirect markers of EIMD.

## 6.5 Conclusion

This study has produced the first data of its kind to indicate that the magnitude of EMD and time-course of recovery after high volume resistance exercise is greater in trained middle-aged males compared to their young counterparts. From a practical standpoint, it is suggested that trained middle-aged males be cognisant of the need for greater recovery time and adopt strategies to account for this. Moreover, resistance training in middle-aged males appears to attenuate the losses in peak power after high volume squatting exercise but does not alter the recovery profile of other indirect markers of muscle damage. Applied practitioners should be mindful of these alterations in trained and untrained middle-aged males and programme training accordingly.

# 7. General discussion

## 7.1 Main findings

*7.1.1 The utility of the FitroDyne to measure acute alterations in muscle function*

Current attempts to measure muscle function during and after exercise are often limited to single-jointed dynamometry (e.g. Dalton et al., 2010). Chapter 3 resolved this issue by determining that, during multi-jointed exercises, the FitroDyne rotary encoder is capable of detecting moderate changes in peak and mean velocity and power during both intra- and inter-day assessments. As such, muscle function was measured using a reliable and ecologically valid tool in the studies described in Chapters 4 to 6. Chapter 4 showed that middle-aged males produce lower barbell velocities than young males. In contrast, Chapter 5 revealed no differences in peak velocity between young and middle-aged resistance trained males during high volume squatting exercise (10 x 10 repetitions at 60% 1RM) despite moderate differences in peak power between the groups. The lack of differences reported in Chapter 5 might be owing to the participants pacing in order to prevent premature fatigue. Indeed, the lower barbell velocities observed for 60% 1RM in Chapter 5, compared to Chapter 4, would support such a notion. It is noteworthy that 60% 1RM demonstrated the lowest difference between groups in Chapter 4, which might not be present during squats performed with multiple repetitions. Furthermore, the differences in peak power during exercise reported in Chapter 5 are likely a result of differences in force and not velocity as previously suggested (Petrella et al., 2005).

At 0, 24 and 72 hours after high volume squatting exercise the trained middle-aged group were subject to greater decrements in peak power at 20 and 80% 1RM than their young counterparts. It is likely that the more substantial losses in peak power in the middle-aged group are in part explained by damage to the contractile machinery

and/or impairments in E-C coupling, as evidenced by more severe reductions in resting twitch torque. The greater losses of peak power compared to isometric force in the middle-aged group is well supported and reflects an elevated energy cost of contraction (Layec et al., 2014) and impairments in cross-bridge cycling (Dalton et al., 2012) with age. Regarding the middle-aged comparison in Chapter 6, the non-resistance trained males were subject to greater decrements in peak power at 24 and 72 hours than their trained counterparts, despite showing a similar magnitude of muscle damage. Interestingly, this finding suggests that the resistance training history of the trained middle-aged males helps maintain peak power after lower-limb muscle damaging exercise.

*7.1.2 Muscle function and training history characteristics of middle-aged trained males*

Little is known about the muscle function (i.e. strength, velocity and power) characteristics of resistance trained middle-aged males and how they compare to their young counterparts. Chapter 4 reported on the load-velocity and lower-power relationships and strength characteristics in young and middle-aged resistance trained males during bench press, squat and bent-over-row exercises. For all exercises and loads, the young group produced higher barbell velocities than their middle-aged counterparts except bench press at 60 to 80% 1RM. Similarly, for all exercises and loads, peak power was greater in the young than the middle-aged males. Interestingly, the differences in peak power, and strength, were greater during squatting exercise than bench press and bent-over-row. This finding reaffirms previous observations of site-specific strength and power losses in the lower-body compared to upper-body (e.g. Candow & Chilibeck, 2005; Noguiera et al., 2013), but it is the first instance of such differences being observed between trained young and middle-aged males.

Middle-aged males should therefore undertake methods to offset such decrements in strength and power, with the correlation analysis in Chapter 4 providing some clarity regarding the approach to adopt. That is, Chapter 4 reported moderate to strong relationships between velocity and 1RM with peak power for all exercises. Whilst these (partial) correlations are supported by previous interventions demonstrating the importance of high-velocity training in older populations (e.g. Ramirez-Campillo et al., 2014; Sayers & Gibson, 2014) they are the first obtained in resistance trained middle-aged males. Consequently, the data from Chapter 4 indicates that middle-aged males should train to increase 1RM and maximal velocity.

Ageing is associated with decreases in training volume (e.g. Ogles & Masters, 2000), meaning the lower resistance training volume in middle-aged compared to young males (Chapter 4 and 6) was not unexpected. Such findings most likely reflect the influence of social factors (e.g. work, family commitments) on training (Ogles & Masters, 2000; Tanaka & Seals, 2008). Interestingly, the studies in Chapters 4 and 5 noted both groups chose resistance training to maintain ‘strength’, but the middle-aged group also focused on this mode of exercise for ‘health’ or ‘fat loss’ purposes. These are novel findings and, given the specific nature of resistance training adaptations (e.g. Buckner et al., 2016), they might help to explain the losses of velocity and power with ageing.

### *7.1.3 Post-exercise alterations in muscle function in young and middle-aged males*

Chapter 5 reported that immediately after high volume squatting exercise, young and middle-aged resistance trained males were subject to comparable decrements in MVC. These losses were mediated by central (decreases in VA) and peripheral (decreases in resting doublet force) alterations, albeit the middle-aged group was

subject to greater decrements in resting doublet force. These early decrements in MVC were related to a higher HR, peak power and volume load during exercise (Chapter 5). This original observation suggests that to understand the exercise dose-fatigue response with resistance training, applied practitioners can monitor HR, peak power and volume load to estimate post-exercise losses in muscle function.

Chapter 6 revealed that at 24 and 72 hours after squatting exercise, decrements in MVC and resting doublet were moderately greater in the trained middle-aged males than their young counterparts. This new finding suggests that after multi-jointed lower-body exercise, middle-aged males, despite regular resistance training, experience a greater magnitude of muscle damage than young males. In Chapter 6 similar impairments in MVC after muscle-damaging exercise in the middle-aged trained and untrained groups were reported, suggesting previous resistance training history does not reduce the susceptibility to muscle damage in middle-aged males. This vulnerability might be owing to a comparable average peak power produced during squatting exercise in the middle-aged groups, that is, the magnitude of damage being related to the workload during exercise, as reported previously (Toft et al., 2002).

## 7.2 Potential limitations

### *7.2.1 Cross-sectional nature of the studies*

The current research opted to use a cross-section of the resistance trained population to assess their responses to resistance exercise. A concern with such a study design is that it cannot allow the researcher to differentiate between cause and effect, but instead determine the association between age groups (Levin, 2006; Mann, 2003). Given the large differences between the age groups in the studies described in

Chapters 4 to 6 (~18 to 21 years), designing a study which could determine the changes in the responses to resistance over ~20 years would be unfeasible.

A further limitation of the cross-sectional nature of the current research, and the convenience sampling adopted, are the differences in biometric characteristics across the groups. Notably, in Chapter 6, the MU group had a higher FFM than the MT group which is unexpected as resistance training should maintain FFM. It is unclear how this might have affected the findings of Chapter 6, but the reader should be mindful of this when interpreting these data.

### *7.2.2 Use of training status among the middle-aged groups*

The secondary aim of the study in Chapter 6 was to compare the magnitude of muscle damage and time course of recovery in trained and untrained middle-aged males and establish how resistance training experience in this group affected the acute response to exercise. Typically, past studies have performed two bouts of muscle-damaging exercise with up to six months between bouts (e.g. Nosaka et al., 2001) to assess the impact of the previous resistance exercise bout on the magnitude of muscle damage in a second bout. However, the current study (Chapter 6) used groups with different resistance training histories and thus did not directly determine the effects of the repeated bout effect. Moreover, as both groups regularly played sport meant that these middle-aged males might be afforded some protection from the eccentric contractions, which might explain why both middle age groups responded in a similar manner after the exercise.

### *7.2.3 Lack of dietary control in Chapters 5 and 6*

In the studies outlined in Chapters 5 and 6, participants were asked to maintain their normal diet and avoid the consumption of ergogenic supplementation throughout the course of the study. Indeed, there is evidence that protein (Buckley et al., 2010; Farup et al., 2014; Jackman, Witard, Jeukendrup & Tipton, 2010; Waldron et al., 2017) and protein-carbohydrate supplementation (Cockburn, Hayes, French, Stevenson, & St Clair Gibson, 2010) can improve recovery from muscle-damaging exercise. Thus, if some of the participants altered their dietary habits they may have been subject to attenuated alterations in the indirect markers of EIMD. To the best of the author's knowledge, participants maintained their normal diet.

### *7.2.4 Reliability of the FitroDyne in different sub-populations*

The reliability data in Chapter 3 was based predominately on young males, all of whom were resistance trained. However, the samples in Chapter 4 to 6 used trained and untrained middle-aged males, for which the reliability of the FitroDyne has not yet been established. Whilst this might be a limitation of Chapters 4 to 6, the data (within-subject errors) was found to be homoscedastic. This would indicate that regardless of the power or velocity values produced (i.e. lower for middle-aged males than young males) the reliability is similar amongst the sample. Moreover, all participants were familiarised until they could produce velocities and powers within a 10% variation (Batterham & George, 2003), meaning any change in muscle function greater than 10% would be deemed true. Regardless, future research might wish to investigate the reliability of the FitroDyne in different populations.

## 7.3 Future directions

### *7.3.1 Acute responses to multi-jointed upper-body exercise in middle-aged males*

As noted in Chapter 2 (see section 2.2) the lower-body appears to undergo greater losses in muscle mass, strength and power with ageing. Consequently, the studies in Chapters 5 and 6 determined the acute responses to lower-body resistance training in young and middle-aged males. Whilst there have been attempts to determine the acute response to upper-body exercise between age groups (e.g. Lavender & Nosaka, 2006a; Lavender & Nosaka, 2006b; Lavender & Nosaka, 2007) these were isolated to single-joint models which do not represent the multi-jointed nature of middle-aged males resistance training habits. Moreover, it is well documented that the upper-body is more susceptible to eccentric exercise than the lower-body (Chen, Lin, Chen, Lin & Nosaka, 2011; Jamurtas et al., 2005; Saka et al., 2009). As such, future studies should determine the acute response to multi-jointed upper-body damaging exercise in middle-aged males.

### *7.3.2 Protein strategies to enhance the recovery of middle-aged males*

Hyperaminoacidemia after protein ingestion stimulates muscle MPS that is thought to enhance recovery from resistance exercise (Buckley et al., 2010; Cockburn et al., 2010; Waldron et al., 2017). It is possible that protein supplementation induces a faster recovery of muscle function after muscle-damaging resistance exercise (Buckley et al., 2010; Cockburn, et al., 2008; Farup et al., 2014; Jackman, Witard, Jeukendrup & Tipton, 2010; Waldron et al., 2017), despite a review by Pasiakos and colleagues disputing this claim (Pasiakos, Lieberman & McLellan, 2014). However, the different study designs and large variability in indirect markers might contribute to the equivocal findings (Pasiakos et al., 2014). Importantly, for MPS to be maximised in older men

(~71 years) higher doses of protein (~40 g) are needed compared to younger males (~20 g, respectively; Yang et al., 2012; Churchward-Venne et al., 2016). No data exists regarding the ability of protein ingestion in ameliorating decrements in muscle function after resistance exercise in resistance trained middle-aged males, which provides a direction for future research.

### *7.3.3 Resistance training to maximise strength and power in middle-aged males*

As detailed in section 2.4.1, resistance training provides a potent method for reducing the age-associated losses in strength and power with ageing. However, these studies are limited to older populations (> 60 years) who do not resistance train (e.g. Newton et al., 2002; Sayers & Gibson, 2014). Future studies should employ methods to optimise strength and power in resistance trained middle-aged males. For example, with the correlations in Chapter 4 in mind, a study that aims to increase both maximum velocity and 1RM (i.e. mixed-methods approach) might optimise adaptations in middle-aged resistance trained males. Chapter 5 also described a dose-fatigue response between certain internal (HR and OMNI-RPE) and external (volume load and peak power) loads and post-exercise decrements in muscle function. Future work might extend these observations by adopting longitudinal designs to report on the dose-response relationship between internal and external loads with resistance training induced gains in strength and power.

### *7.3.4 Acute strength and power responses to resistance exercise in females*

This thesis focused on males due to the occurrence of more severe loss muscle mass, strength and power with age than their female counterparts (e.g. Abe et al., 2011). As such the current data can only be applied to males. Moreover, there is evidence to

suggest that the acute responses to resistance exercise are different in males and females (e.g. Christie et al., 2011; Kendall & Eston, 2002). Consequently, future research may seek to replicate the study designs in Chapters 4 to 6 using a female sample, or perhaps provide a middle-aged male and female comparison.

# 8. Conclusions

This thesis sought to compare the acute strength and power responses to resistance exercise among young and middle-aged resistance trained males. Chapter 3 determined that, in a sample of young resistance trained males, both intra- and inter-day, the FitroDyne could detect moderate, but not small, changes in peak and mean velocity and power across bench press, squat and bent-over row. Notably, there were some concerns regarding the intra-day reliability of bench press at 80% 1RM that were alleviated inter-day. Practically, when assessing muscle function the FitroDyne provides an alternative to the use of single-jointed isometric and isokinetic dynamometry.

Chapter 4 provided a comprehensive analysis of the load-velocity and load-power profiles during bench press, squat and bent-over row in young and middle-aged resistance trained males. Despite their regular resistance training, middle-aged males were unable to achieve high barbell velocities at low external loads and peak powers at all external loads during all three exercises. The strong relationships between strength and velocity with peak power in the middle-aged group suggest that athletes should undertake specific methods to improve these components.

The findings from Chapter 5 highlighted that internal (HR, RPE and sRPE), but certain external (peak power and volume load) load responses can be monitored during squatting exercise in a like manner among young and middle-aged males. Moreover, squatting exercise impairs peak power to a greater extent that isometric force in middle-aged males compared to their young counterparts. The correlations observed in Chapter 5 indicate that certain internal (HR and OMNI-RPE) and external (mean peak power and volume load) loads are positively related to the post-exercise

decrements in muscle function. As such, the applied practitioner should monitor these variables when post-exercise decrements in muscle function are undesirable.

In the final study of this thesis (Chapter 6), it emerged that, compared to the young males, trained middle-aged males were subject to greater symptoms of muscle-damage and an impaired recovery profile. Consequently, trained middle-aged males should adopt strategies to enhance their recovery profile. In addition, resistance training experience in middle-aged males appears to attenuate the loss of peak power after squatting exercise.

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# Appendices

## Appendix A

## Ethical approval letter for Chapters 3 and 4

***Faculty of Life Sciences***
***Research Ethics Committee***
frec@chester.ac.uk

John Fernandes
6 Walpole Street
Chester
CH1 4AG

3rd March 2014

Dear John,

**Study title:** **The intra- and inter-day reliability of the FitroDyne for assessing upper and lower-body muscle function in male athletes.**
**FREC reference:** **871/14/JF/SES**
**Version number:** **1**

Thank you for sending your application to the Faculty of Life Sciences Research Ethics Committee for review.

I am pleased to confirm ethical approval for the above research, provided that you comply with the conditions set out in the attached document, and adhere to the processes described in your application form and supporting documentation.

However, the Committee would like to request the following amendments:-

- On the Participant Information Sheet, 'What will happen to me if I take part?' replace the word 'session' with 'sessions' in the last paragraph and specify the number hours required for abstention from exercise, leading up to testing.
- Consider shortening the Pre-test Health Questionnaire for the individual sessions, focusing on that specific time i.e. immediate illness not apparent at the initial screen.

Please forward amended electronic copies of the documentation to frec@chester.ac.uk

The final list of documents reviewed and approved by the Committee is as follows:

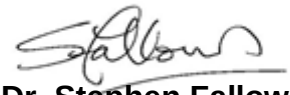
| Document | Version | Date |
|---|---|---|
| Application Form | 1 | January 2014 |
| Appendix 1 – List of References | 1 | January 2014 |
| Appendix 2 – C.V. for Lead Researcher | 1 | January 2014 |
| Appendix 3 – Participant Information Sheet | 1 | January 2014 |
| Appendix 4 – Participant Consent Form | 1 | January 2014 |

| | | |
|---|---|---|
| Appendix 5 – Schematic of Study Design | 1 | January 2014 |
| Appendix 6 – Risk Assessment Form | 1 | January 2014 |
| Appendix 7 – Pre-test Health Questionnaire | 1 | January 2014 |
| Appendix 8 – Email Letter of Invitation | 1 | January 2014 |
| Response to FREC request for further information or clarification | | |
| Appendix 3 – Participant Information Sheet | 2 | February 2014 |
| Appendix 4 – Participant Consent Form | 2 | February 2014 |
| Appendix 5 – Schematic of Study Design | 2 | February 2014 |
| Appendix 7 – Pre-test Health Questionnaire | 2 | February 2014 |

Please note that this approval is given in accordance with the requirements of English law only. For research taking place wholly or partly within other jurisdictions (including Wales, Scotland and Northern Ireland), you should seek further advice from the Committee Chair / Secretary or the Research and Knowledge Transfer Office and may need additional approval from the appropriate agencies in the country (or countries) in which the research will take place.

With the Committee's best wishes for the success of this project.

Yours sincerely,



**Dr. Stephen Fallows**
Chair, Faculty Research Ethics Committee

Enclosures: Standard conditions of approval.
Cc. Supervisor/FREC Representative

**Appendix B**

**Ethical approval for Chapters 5 and 6**

***Faculty of Science and Engineering***
***Research Ethics Committee***

John Fernandes
1 Thomas Brassey Close,
Chester,
CH2 3AE

5th August 2016

Dear John,

**Study title: The time course to recovery from resistance training in young, veteran athletes and veteran non-athletes**
**FSE-REC reference: 063/16/JF/SES**
**Version number: 1**

Thank you for sending your application to the Faculty of Science and Engineering Research Ethics Committee for review.

I am pleased to confirm ethical approval for the above research, provided that you comply with the conditions set out in the attached document, and adhere to the processes described in your application form and supporting documentation.

The final list of documents reviewed and approved by the Committee is as per your response dated Monday 4th July 2016.

Please note that this approval is given in accordance with the requirements of English law only. For research taking place wholly or partly within other jurisdictions (including Wales, Scotland and Northern Ireland), you should seek further advice from the Committee Chair / Secretary or the Research and Knowledge Transfer Office and may need additional approval from the appropriate agencies in the country (or countries) in which the research will take place.

With the Committee's best wishes for the success of this project.

Yours sincerely,

---

**Eustace Johnson**
Chair, Faculty of Science and Engineering Research Ethics Committee

Enclosures: Response to FSE-REC template

C.c. Supervisor FSE-REC Representative

## Appendix C

## Participant information sheet for Chapter 3

### The intra- and inter-day reliability of the FitroDyne for assessing upper and lower-body muscle function in male athletes

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask the researcher if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this.

**What is the purpose of the study?**
Despite resistance training being performed by athletes of all ages, ageing results in changes in muscle quality (i.e. changes in body composition) that leads to reductions in muscle performance. Accordingly, it is important to reliably quantify muscle function during appropriate exercises across a range of ages to understand the factors that influence resistance training performance.

Assessing muscle performance during resistance exercise using a rotary encoder might provide a useful measure to quantify muscle performance. However, no study has yet assessed the reliability of this apparatus when used with such exercises, or across a range of ages. The primary purpose of this study is to determine the intra- and inter-day reliability of the FitroDyne rotary encoder for assessing upper and lower-body muscle function in male athletes. Secondly, the study will seek to examine differences in muscle performance between younger (< 35 years) and older (35+ years) athletes during typical resistance exercises.

**Why have I been chosen?**
You have been selected because you are male that has experience in resistance training.

**Do I have to take part?**
It is up to you to decide whether or not to take part. If you decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect you in any way.

**What will happen to me if I take part?**
You will be asked to complete an informed consent form and a health questionnaire. After this you will attend the Strength and Conditioning laboratory for testing on four occasions (see attached flow chart). The initial sessions will consist of an estimation of your one repetition maximum (1RM) from a 5 repetition maximum on bench press and squat, a 1RM on bench pull exercise, and assessment of body composition using skinfold callipers. Thereafter, you will complete three more sessions on two days (at 24 and 48 hours, respectively; see attached flow chart) in which you will perform three

repetitions of bench press, squat and bench pull exercise at 20, 30, 40, 50, 60, 70 and 80% 1RM. All session will last no more than 1.5 hours except trial 2 which will be two 1.5 hour session with a 2 hour break between trials (total 5 hours). Please note that you will be required to maintain your normal schedule but abstain from exercise in the hours leading up to testing.

**What are the possible disadvantages and risks of taking part?**
There may be some risk of muscle strain or injury when conducting testing. However, you have experience in resistance training and the researcher is a Personal Trainer who will assist with technique and spotting where needed.

**What are the possible benefits of taking part?**
The potential benefits of taking part include an assessment of your body composition. You will also have a full muscle functional profile assessment during bench press, squat and bench pull movements. Should you also want information on how to improve either body composition or muscle function the researcher can provide this.

**What if something goes wrong?**
If you wish to complain or have any concerns about any aspect of the way you have been approached or treated during the course of this study, please contact Professor Sarah Andrew, Dean of the Faculty of Life Sciences, University of Chester, Parkgate Road, Chester, CH1 4BJ, 01244 513055.

**Will my taking part in the study be kept confidential?**
All information which is collected about you during the course of the research will be kept strictly confidential so that only the researcher carrying out the research will have access to such information.

**What will happen to the results of the research study?**
The results will be written up into a thesis for the researcher's PhD. Individuals who participate will not be identified in any subsequent report or publication.

**Who is organising the research?**
The research is conducted as part of a PhD in Exercise Physiology within the Department of Sport and Exercise Sciences at the University of Chester. The study is organised with supervision from the department, by John Fernandes, a PhD student.

**Who may I contact for further information?**
If you would like more information about the research before you decide whether or not you would be willing to take part, please contact:

*John Fernandes (j.fernandes@chester.ac.uk)*
01244 513426
**Thank you for your interest in this research.**

## Appendix D

## Participant information sheet for Chapter 4

### A comparison of muscle function in young and veteran male athletes during upper and lower-body exercises.

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask the researcher if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this.

**What is the purpose of the study?**
Despite resistance training being performed by athletes of all ages, ageing results in changes in muscle quality (i.e. changes in body composition) that leads to reductions in muscle performance. Accordingly, it is important to quantify muscle function during appropriate exercises across a range of ages to understand the factors that influence resistance training performance. The purpose of this study is to examine differences in muscle performance between younger (< 35 years) and older (35+ years) athletes during typical resistance exercises.

**Why have I been chosen?**
You have been selected because you are male that has experience in resistance training.

**Do I have to take part?**
It is up to you to decide whether or not to take part. If you decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect you in any way.

**What will happen to me if I take part?**
You will be asked to complete an informed consent form and a health questionnaire. After this you will attend the Strength and Conditioning laboratory for testing on two occasions. The initial sessions will consist of an estimation of your one repetition maximum (1RM) from a 5 repetition maximum on squat and a 1RM on bench press and bent-over-row exercise, and assessment of body composition using skinfold callipers. Thereafter, you will complete one session (minimum 48 hours later) in which you will perform three repetitions of bench press, squat and bench pull exercise at 20, 30, 40, 50, 60, 70 and 80% 1RM. All session will last no more than 1.5 hours. Please note that you will be required to maintain your normal schedule but abstain from exercise in the hours leading up to testing.

**What are the possible disadvantages and risks of taking part?**

There may be some risk of muscle strain or injury when conducting testing. However, you have experience in resistance training and the researcher is a Personal Trainer who will assist with technique and spotting where needed.

**What are the possible benefits of taking part?**
The potential benefits of taking part include an assessment of your body composition. You will also have a full muscle functional profile assessment during bench press, squat and bench pull movements. Should you also want information on how to improve either body composition or muscle function the researcher can provide this.

**What if something goes wrong?**
If you wish to complain or have any concerns about any aspect of the way you have been approached or treated during the course of this study, please contact Professor Sarah Andrew, Dean of the Faculty of Life Sciences, University of Chester, Parkgate Road, Chester, CH1 4BJ, 01244 513055.

**Will my taking part in the study be kept confidential?**
All information which is collected about you during the course of the research will be kept strictly confidential so that only the researcher carrying out the research will have access to such information.

**What will happen to the results of the research study?**
The results will be written up into a thesis for the researcher's PhD. Individuals who participate will not be identified in any subsequent report or publication.

**Who is organising the research?**
The research is conducted as part of a PhD in Exercise Physiology within the Department of Sport and Exercise Sciences at the University of Chester. The study is organised with supervision from the department, by John Fernandes, a PhD student.

**Who may I contact for further information?**
If you would like more information about the research before you decide whether or not you would be willing to take part, please contact:

*John Fernandes (j.fernandes@chester.ac.uk)*
01244 511988
**Thank you for your interest in this research.**

## Appendix E

### Participant information sheet for Chapter 5 and 6

### The time course to recovery from resistance training in young, veteran athletes and veteran non-athletes

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask the researcher if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this.

**What is the purpose of the study?**
The purpose of this study is to compare the internal and external load during resistance training in young and veteran athletes and non-trained veterans. A secondary aim is to determine the time course to recovery in young and veteran male resistance trained athletes and non-trained veterans.

**Why have I been chosen?**
You have been selected because you are a male that partakes in sport and regularly resistance trains and/or can proficiently squat.

**Do I have to take part?**
It is up to you to decide whether or not to take part. If you decide to take part, you will be given this information sheet to keep and be asked to sign a consent form. You are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect you in any way.

**What will happen to me if I take part?**
You will be asked to complete an informed consent form and a health questionnaire. After this you will attend the Strength and Conditioning Laboratory for testing on four occasions (see attached flow chart). The initial sessions will consist of an estimation of your one repetition maximum (1RM) back squat from a three repetition maximum test, an assessment of body composition using skinfold callipers, and familiarisation to maximal voluntary contractions, lower-limb power output and muscle soreness.

Two to four days later you will return to the laboratory for baseline measurements (lower-limb power output, voluntary activation during isometric knee extension, muscle soreness and creatine kinase activity from a fingertip blood sample) and a muscle-damaging exercise bout comprising 10 x 10 squats at 60% 1RM (with 120s rest between sets). You will be required to provide your perceived exertion at the end of each set and at the end of the exercise bout. Repeated measures (lower-limb power output, voluntary activation during isometric knee extension, muscle soreness and creatine kinase activity) will be recorded 0, 24 and 72 hours after the initial exercise bout. The sessions will last no more than 60 minutes except the second session, which will last 2 hours. Please note that you will be required to abstain from exercise during the course of this schedule.

**What are the possible disadvantages and risks of taking part?**
There might be some risk of muscle damage, strain or injury when conducting testing. However, you have experience in resistance training and the researcher is a Personal Trainer who will assist with technique and spotting where needed. Furthermore, you will experience a short bout of muscle soreness as a consequence of the resistance exercise. This will be most evident at 48 hours after which the symptoms will subside and have completely disappeared by one week. These symptoms have no lasting effect and are common in all exercising populations.

**What are the possible benefits of taking part?**
The potential benefits of taking part include an assessment of your body composition and lower-body power and strength. Additionally, the bout of resistance exercise is likely to afford some protection from further symptoms of muscle damage to your lower-body muscle for a short period of time (up to 6 months) afterwards.

**What if something goes wrong?**
If you wish to complain or have any concerns about any aspect of the way you have been approached or treated during the course of this study, please contact the Dean of the Faculty of Science and Engineering, University of Chester, Parkgate Road, Chester, CH1 4BJ, 01244 513923.

**Will my taking part in the study be kept confidential?**
All information that is collected about you during the course of the research will be kept strictly confidential so that only the researcher carrying out the research will have access to such information.

**What will happen to the results of the research study?**
The results will be written up into a thesis for the researcher's PhD. Individuals who participate will not be identified in any subsequent report or publication.

**Who is organising the research?**
The research is conducted as part of a PhD in Exercise Physiology within the Department of Sport and Exercise Sciences at the University of Chester. The study is organised with supervision from the department, by John Fernandes, a PhD student.

**Who may I contact for further information?**
If you would like more information about the research before you decide whether or not you would be willing to take part, please contact:

*John Fernandes (j.fernandes@chester.ac.uk)*
01244 511988

**Thank you for your interest in this research.**

**Appendix F**
**Example health questionnaire**

## PRE-TEST HEALTH QUESTIONNAIRE

(PLEASE NOTE THAT THIS INFORMATION WILL BE CONFIDENTIAL)

Name:……. DOB:…….. Age:……

Blood Pressure:..... Heart rate:.....

Project Title: *The time course to recovery from resistance training in young and veteran athletes*

Please answer these questions truthfully and completely. The purpose of this questionnaire is to ensure that you are fit and healthy enough to participate in this laboratory practical/research project.

| | **Yes** | **No** |
|---|---|---|
| 1. Have you in the past suffered from a serious illness or accident. | ☐ | ☐ |

If Yes, please provide details

……
……

| | **Yes** | **No** |
|---|---|---|
| 2. Have you consulted your doctor the last 6 months | ☐ | ☐ |

If Yes, please provide details

……
……

3. Do you suffer, or have you suffered from:

| | Yes | No |
|---|---|---|
| Asthma | ☐ | ☐ |
| Diabetes | ☐ | ☐ |
| Bronchitis | ☐ | ☐ |
| Epilepsy | ☐ | ☐ |
| High blood pressure | ☐ | ☐ |
| Haemorrhoids | ☐ | ☐ |

| | **Yes** | **No** |
|---|---|---|
| 4. Is there any history of heart disease in your family | ☐ | ☐ |

| | **Yes** | **No** |
|---|---|---|
| 5. Are you suffering from any infectious skin diseases, sores, cuts or open wounds, or infections i.e., Hepatitis B, HIV, etc.? | ☐ | ☐ |

If Yes, please provide brief details

……
……

| | Yes | No |
|---|---|---|
| 6. Are you currently taking any medication | ☐ | ☐ |

If Yes, please provide details

……
……
……

| | Yes | No |
|---|---|---|
| 7. Are you suffering from a disease that inhibits the sweating process | ☐ | ☐ |

| | Yes | No |
|---|---|---|
| 8. Is there anything to your knowledge that may prevent you from participating in the testing that has been outlined to you? | ☐ | ☐ |

If Yes, please provide details

……
……
……

**Your Recent Condition**

| | Yes | No |
|---|---|---|
| • Have you eaten in the last 2 hours? | ☐ | ☐ |

If Yes, please provide details

……
……

| | | |
|---|---|---|
| • Have you consumed alcohol in the last 24hr | ☐ | ☐ |
| • Evaluate your diet over the last two days. | **Poor Average Good Excellent** | |
| • Have you had any kind of illness or infection in the last 2 weeks | ☐ | ☐ |
| • Have you exercised in the last 2 days? | ☐ | ☐ |

If Yes, please describe below

……
……

Persons will not be permitted to take part in any experimental testing if they:

- have a known history of medical disorders (i.e. hypertension, heart or lung disease)
- have a fever, suffer from fainting or dizzy spells
- are currently unable to train because of a joint or muscle injury
- have had any thermoregulatory disorder
- have gastrointestinal disorder
- have a history of infectious diseases (i.e. HIV or Hepatitis B)
- have, if pertinent to the study, a known history of rectal bleeding, anal fissures, haemorrhoids or any other similar rectal disorder.

My responses to the above questions are true to the best of my knowledge and I am assured that they will be held in the strictest confidence.

Name: (Participant)…… Date:……..

Signed (Participant): …….

Name: (Researcher): John Fernandes Date:…….

Signed (Researcher): …….

**Appendix G**
**Example informed consent**

**Title of Project:** *The time course to recovery from resistance training in young and veteran athletes*

**Name of Researcher:** *John Fernandes*

**Name of Supervisors:** *Professor Kevin Lamb and Professor Craig Twist*

Please initial box

1. I confirm that I have read and understand the information sheet for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason and without my legal rights being affected. ☐

3. I agree to take part in the above study. ☐

| _____ | _____ | _____ |
|---|---|---|
| Name of Participant | Date | Signature |
| John Fernandes | | |
| _____ | _____ | _____ |
| Researcher | Date | Signature |

1 for participant; 1 for researcher